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ROLE OF ORIENTATION REFERENCE SELECTION IN MOTION SICKNESS

Semiannual Status Report
NAG 9-117, Supplement 5

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December 1988

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(NASA-CR-184609) ROLE OF ORIENTATION REFERENCE SELECTION IN MOTION SICKNESS Semiannual Status Report (Good Samaritan Hospital and Medical Center) 82 p CSCL 06E G3/52

N89-15513

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SUMMARY OF PROPOSAL

Previous experiments with moving platform posturography have shown that different people have varying abilities to resolve conflicts among vestibular, visual, and proprioceptive sensory signals used to control upright posture. In particular, there is one class of subjects with a vestibular disorder known as benign paroxysmal positional vertigo (BPPV) who often are particularly sensitive to inaccurate visual information. That is, they will use visual sensory information for the control of their posture even when that visual information is inaccurate and is in conflict with accurate proprioceptive and vestibular sensory signals. BPPV has been associated with disorders of both posterior semicircular canal function and possibly otolith function. The present proposal hopes to take advantage of the similarities between the space motion sickness problem and the sensory orientation reference selection problems associated with the BPPV syndrome. These similarities include both etiology related to abnormal vertical canal-otolith function, and motion sickness initiating events provoked by pitch and roll head movements.

The objectives of this proposal are to explore and quantify the orientation reference selection abilities of subjects and the relation of this selection to motion sickness in humans. The overall objectives of this proposal are to determine (1) if motion sickness susceptibility is related to sensory orientation reference selection abilities of subjects, (2) if abnormal vertical canal-otolith function is the source of abnormal posture control strategies and if it can be quantified by vestibular and oculomotor reflex measurements, and (3) if quantifiable measures of perception of vestibular and visual motion cues can be related to motion sickness susceptibility and to orientation reference selection ability.

SUMMARY OF PROJECT STATUS

Three test devices are required for the proposed experiments. They are (1) moving posture platform, (2) servo controlled vertical axis rotation chair with an independently controllable optokinetic stimulator, and (3) a two-axis rotation chair for the generation of pitch and roll motions. The first two devices are currently functional and are routinely used for both clinical and research testing. The moving posture platform software has recently been updated in order to provide a flexible method for quickly designing new experimental protocols. Work is continuing on the two-axis rotation device. The development of this two-axis rotator has been a major focus in the first two years and will be described in more detail below.

Experiments involving the perceptual feedback technique developed by Zacharias and Young (Exp Brain Res, 41:159-171, 1981) have continued. We have increased the number of experimental subjects on a set of experiments designed to look at the correlation between vestibulo-ocular reflex parameters and the perception of rotation. We have also conducted experiments exploring the effect of time-delayed visual feedback on the perception of rotation, and the effect of time-delayed visual and proprioceptive feedback on posture control.

TWO-AXIS ROTATOR DEVELOPMENT

The two-axis rotator is a versatile, general purpose stimulator for vestibular and visual-vestibular interaction studies. It consists of two gimbals powered

by rotary hydraulic actuators. The inner gimbal produces yaw axis rotations of the subject. The outer gimbal rotates the subject about a horizontal axis which passes through the subject's ears. Figure 1 shows a line drawing of the device.

We have been working on 6 projects related to the two-axis rotator in the past 6 months. These are (1) achieving acceptable performance of pitch axis motions, (2) construction of a chair/restraint system for subjects, (3) design and construction of a optokinetic (OK) projector and projection screen, (4) construction of a DC servo controlled torque motor which will be interchangeable with either the yaw or pitch axis hydraulic actuators, (5) development of a data collection and stimulus delivery program, and (6) construction of an on-board electronics module.

(1) Pitch Axis Hydraulic Actuator

The control of the pitch axis movement has been a problem since the initial installation of the two-axis rotator. Specifically the dynamic response characteristics have been poor. Neither adjustments in the feedback controller nor a larger capacity servovalve and accumulator to stabilize hydraulic pressure fluctuations were not able to fix the problem. Since the design equations indicated that the pitch axis should be controllable, we looked for another reason for the poor control and found it in a defective mechanical coupling between the actuator and its shaft. The actuator and housing were sent back to the manufacturer for repair and for some other modifications. We have received the repaired item and reinstalled it. Testing has shown that the movement control has improved significantly, and we are satisfied with its present performance.

(2) Chair/Restraint System

The object of the chair/restraint system is to hold the subject firmly and comfortably in position during motion of the two-axis rotator. The test subject sits in a kneeling position with his body held in a chair assembly. The chair assembly is nearly completed, and consists of a fiber glass race car seat mounted in an aluminum frame. The frame sits on a jack within the inner gimbal. The jack is used to raise and lower the seat. The seat is supported from behind by two cross bars which ride up and down on two stainless steel tubes. Locking mechanisms on the tubes and cross bars allow for chair adjustment forward and back, left and right, and up and down. This adjustment assures that the subject's head can be positioned at the center of rotation of the two-axis gimbals, and allows for some degree of off-axis positioning of the head. A second jack mechanism and slider assembly in the base of the chair is used to position the angle of the subject's knees and then to restrain the knees in order to stabilize the lower body. The two projects which remain for chair completion are a head holder for the chair, and a handle bar hold with proximity switches for emergency stopping of a test by the subject.

(3) Optokinetic Projector

The optokinetic projector provides a full field visual stimulus to a subject seated in the two-axis rotator. Visual field rotation is controlled by a small torque motor which drives a slit projector through gear reduction belts. The compact projector can be mounted either above or to the side of the subject's

head. Mounting the projector above the head provides a visual stimulus moving about the subject's yaw axis. Mounting to the side of the head provides a vertical OK stimulus. Two different cylindrical screens, one horizontal and one vertical, provide the surface on which the images of stripes are projected. Some problems with the smoothness of the belt drive and the quality of the projected light stripes have been overcome. The mounting systems for the vertical and horizontal axes projection screens to the two-axis rotator have both been completed.

(4) Electric Torque Motor

There is a class of experiments where sustained rotations at constant velocities have proven useful in identifying vestibular reflex responses related to otolith function. These experiments involve rotating a subject at constant velocity about an axis tilted off of earth vertical. Subjects produce sustained horizontal eye movements under these conditions. Animal experiments have shown that these eye movements are the result of central processing of otolith responses.

Since this grant is interested in otolith function, and sensory integration of otolith, semicircular canal, and visual motion information, we designed a DC torque motor system which would permit constant velocity or very low frequency rotational movements. The motor will be interchangeable with the hydraulic actuators that are currently in the two-axis rotator.

The preliminary design of the motor housing was completed last year, and the design was finalized in January 1988 after receiving the Inland motor. Construction of the housing was started at that time, was completed in mid May 1988, and was then delivered. The motor is designed to have 120 ft-lb of torque, and will be able to deliver that torque at a peak velocity of 320 deg/sec. Higher velocities will be possible at reduced torques.

The power supply, transformer, and servo controller necessary to drive the motor were received from Inland Motor Corp. We have completed the wiring and integration of these components. During testing and calibration of the motor, we discovered mechanical motor alignment problems. The motor was sent back to the manufacturer for correction. We have received the repaired motor and verified that the problem has been fixed.

(5) Data Collection and Stimulus Delivery Program

To study the eye movements evoked by vestibular and visual system reflexes in our two-axis rotator it will be necessary to record the eye movements in relationship to the stimuli which cause them. In order to do this the exact time synchrony between the analog and video signals must be preserved. We have made substantial progress on a Modula-2 program which synchronizes this data collection and stimulus delivery timing with the timing of the video recording of the eye movements. This program allows the definition of arbitrary three axis motion profile (yaw, pitch, optokinetic) including ramp, sine, square wave, and constant positioning.

(6) On-board Electronics Module

An on-board electronics module is needed in order to record various physiological signals during testing on the two-axis rotator. The electronics

module has recently been completed. It resides on the yaw axis of the two-axis rotator, and contains two channel EOG electronics, video camera electronics, and a pulsed power supply for the infrared video camera light source.

PERCEPTUAL FEEDBACK EXPERIMENTS

In 1981, Zacharias and Young presented a method which allowed for the quantification of a subject's perception of rotation under the combined influence of visual and vestibular cues. In this technique, the subject has control over the rotational motion of the chair by adjusting a potentiometer. Subjects are seated in the vertical axis rotation test room with the potentiometer mounted on the arm of the chair. The output of this potentiometer is summed with a velocity command signal from a computer and this summed signal is delivered to the velocity command input of the chair's servo motor. The goal of the subject is to continuously adjust the potentiometer so that he feels like he is not moving. A "perfect" subject would be able to hold himself stationary in space by adjusting the potentiometer so that its output was equal but opposite to the computer's command signal. "Real" subjects do not remain stationary because of the dynamics of their motion perception and motor reaction systems, and because of presumed imbalances in the vestibular receptors.

The rotation of the subject's chair and the visual surround can be independently manipulated. We have used 8 different sensory environments for our preliminary experiments. These include (1) chair rotation in the dark, (2) rotation of the visual surround with the chair stationary, (3) chair rotation with the visual surround velocity equal to the chair velocity, (4) chair rotation with a constant velocity surround, (5) chair rotation with the velocity of the visual surround equal to the chair velocity plus a constant, (6) chair rotation with a stationary visual surround, (7) chair rotation with a time delayed, earth-referenced visual surround, and (8) chair rotation with a time delayed, subject-referenced visual surround. Condition (2) is used to test the motor control dynamics of the subject. Conditions (1) and (3)-(8) represent a variety of sensory environments in which the subject is forced to deal with either accurate, inaccurate, conflicting, or absent sensory cues about his motion. Test conditions 7 and 8 add a time delay between chair rotation and the rotation of the visual surround. Time delays are of interest because of their potential to disrupt the coordination of reflexes associated with orientation control.

A total of 21 subjects have been tested in a protocol which includes tests of horizontal VOR function as well as the eight perceptual feedback tests. The goal of these preliminary experiments was to (1) gain experience with this technique, (2) verify the results of Zacharias and Young, (3) extend the results of Zacharias and Young by including a wider variety of sensory environments, (4) determine the consistency of results for individual subjects tested over time, and (5) look for correlations between VOR and perceptual feedback test results. These techniques will later be extended to the two-axis rotator, compared with results of moving platform posturography, and correlated with the results of vestibular and oculomotor reflex measurements and measurements of motion sickness susceptibility.

Repeatability and Correlation with VOR Tests

We have increased the number of test subjects using this protocol from 17 reported in the previous progress report to 21 in order to verify some of the correlations which were emerging. Some of the data on the 21 subjects tested has been analyzed, and has been presented at a poster session at the American Society for Gravitational and Space Biology's fourth annual meeting in Washington, D.C. in October 1988 (see the "Paper Submission and Poster Session" section of this report, and abstract and poster copy attached.)

The article by Zacharias and Young suggested that the drift of the subject during rotation in the dark, or with subject-referenced vision, might be due to an imbalance in the encoded motion information coming from the two halves of the vestibular system in opposite ears. This is also the interpretation which is generally given to the presence of bias, or directional preponderance observed in tests of horizontal VOR function. We anticipated that there might be a correlation between the drift observed in perceptual feedback tests and the bias recorded in VOR tests. However we have not found any obvious correlation between these two measures in the subjects tested to date. It may be possible that normal subjects have too small a range of bias and drift to provide a reliable correlation. However the bias measured for a given subject does appear to remain consistent over time, as does drift. That is, the reliability of the measurement of these two parameters seems to be fairly good. This would argue that the lack of correlation between these two measures is real, and not an artifact of their limited range, at least in normal subjects. This observation appears to be in agreement with some other data in the literature (see for example Guedry in Handbook of Sensory Physiology, Springer-Verlag, 1974) which has identified differences between the dynamics of reflex responses and perception. We believe that exploring these differences and their possible association with motion sickness may be productive.

Effect of Visual Feedback Time Delay on Rotation Perception

We have collected some preliminary data from perceptual feedback experiments in which the visual motion cues are provided to the subject, but are delayed in time by a variable amount. For example, when a subject rotates to the right, normally the world moves to his left in synchrony with his movement. Introducing a time delay into this situation, when the subject moves to the right, the world initially moves to the right with him, but after a specified interval, or time delay, the world begins to move to his left in a normal manner. We refer to this as an earth-referenced time delay.

Another situation occurs when the visual field moves with the subject so that the visual field does not give any cue about the subject's motion. We refer to this as a subject-referenced visual environment. When a time delay is introduced into this situation, motion of the subject to the right is initially accompanied by motion of the visual surround to the left with respect to the subject. However if the subject continues to rotate to the right, after the time delay interval, the visual surround will start to rotate with the subject.

The motivation for these time-delayed feedback experiments came from some unpublished observations by L.M. Nashner on posture control. He observed that the posture control of some normal subjects were greatly affected by the presence of a time-delayed movement of the visual surround. His experiments

were of a form equivalent to our subject-referenced time delays. Our results using the perceptual feedback technique have not been particularly compelling, and apparently do not agree with Nashner's results. There have not been any reports of disorientation at any of the time delays so far tested which might correspond to the disruption of posture control reported by Nashner. We have increased the number of test subjects using subject-referenced time delay stimulus from 3 reported in the previous progress report to 6 in order to test the hint of poorer control of rotation at certain time delays. Looking at the additional data, no significant control difference was found at any of the time delays tested.

We have repeated Nashner's experiments on the moving posture platform in order to verify his earlier observations. Since posture control involve the otolith organs, vertical semicircular canals, and proprioceptive systems, it presents quite a different sensory environment than our vertical-axis rotations, and we thought this might account for the apparently different findings. We tested 27 subjects, 16 "normal" and 11 "abnormal" subjects, and found no significant loss of posture control on any of the time delays tested. If we had been able to verify Nashner's results, and confirm that they differ from our vertical-axis tests, we would have been able to pursue these time delay experiments on the two-axis rotation device once it was functional.

PAPER SUBMISSION AND POSTER SESSION

We have recently submitted two papers for publication to Experimental Brain Research: "Age-related Changes in Human Vestibulo-ocular and Oculomotor Reflexes" by R.J. Peterka, F.O. Black and M.B. Schoenhoff, and "Age-related Changes in Human Posture Control" by R.J. Peterka and F.O. Black. These papers report the response properties of horizontal vestibulo-ocular reflex, optokinetic reflex and posture control in 216 human subjects ranging in age from 7 to 81 years. The two papers are very closely connected in that the same experimental subjects participated in both studies. Copies of both papers are enclosed.

The PI attended the American Society for Gravitational and Space Biology's fourth annual meeting in Washington, D.C. on October 20-23, 1988 and displayed a poster titled "Is Inaccurate Perception of Vertical Axis Rotation related to Asymmetrical Vestibulo-ocular Reflex Function in Normal Human Subjects?" by R.J. Peterka and M.S. Benolken. This poster summarized the latest analysis of our perceptual feedback experiments. A copy of the abstract and poster is attached. We are presently working on a paper from this abstract to be submitted to Aviation, Space, and Environmental Medicine.

IS INACCURATE PERCEPTION OF VERTICAL AXIS ROTATION RELATED TO ASYMMETRICAL VESTIBULO-OCULAR REFLEX FUNCTION IN NORMAL HUMAN SUBJECTS?

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Subjects seated in a vertical axis rotation chair in the dark controlled their rotational velocity with a potentiometer. Their goal was to null out pseudorandom rotational perturbations introduced by the experimenter in order to remain perceptually stationary. Most subjects showed a slow linear drift of velocity to one side. Zacharias and Young (Exp Brain Res 41:159-171, 1981) proposed a model which accounted for their similar experimental results. The model postulated that the source of the drift was an imbalance in vestibular function between the two ears. Since it is often assumed that the small deviations from perfect vestibulo-ocular reflex (VOR) symmetry are also related to an imbalance between the two ears, we looked for correlations between drift measured in these perceptual feedback experiments and several measures of VOR symmetry. No correlations were found. Measurement errors could not account for these poor correlations since repeated tests in the same subjects of both perceptual drift and VOR symmetry were highly correlated. We concluded that a simple imbalance between the two ears cannot account for both observations.

Supported by NASA grant NAG 9-117

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INTRODUCTION

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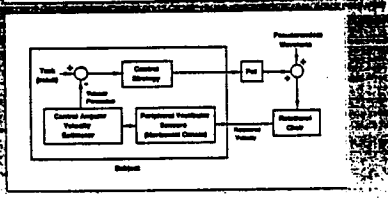
Zacharias and Young (Exp Brain Res 41:159-171, 1981) previously observed that many subjects show a directional bias in the perception of horizontal plane body motion. They postulated that the source of this perceptual bias was an imbalance between the signals from the horizontal semicircular canals of the two ears.

It is often assumed that deviations from perfect vestibulo-ocular reflex (VOR) symmetry are also related to an imbalance between the two ears. VOR asymmetries and perceptual bias should be related if an ear imbalance is the source for both.

METHODS

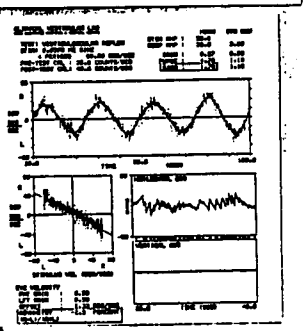
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Perceptual Feedback Tests. Subjects seated in a vertical axis rotational chair in the dark controlled their rotational velocity with a potentiometer. Their task was to null out pseudorandom rotational perturbations introduced by the experimenter in order to remain perceptually stationary. We used the average drift of rotational chair velocity away from zero as a measure of perceptual bias.



3

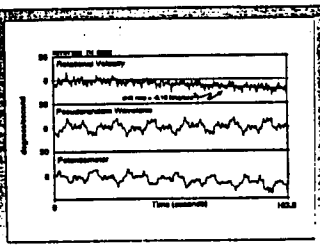
VOR Tests. We quantified VOR responses to sinusoidal vertical axis rotations at 0.05, 0.2 and 0.8 Hz by comparing horizontal slow phase eye velocity to chair velocity. One VOR parameter related to response symmetry is VOR bias, which is a measure of the average slow phase eye velocity over an integer number of cycles. Another parameter is VOR ASYMMETRY which compares the VOR gains for right and left rotations. We measured VOR parameters before and after a set of 12 perceptual feedback tests which included 3 rotations in the dark.



RESULTS

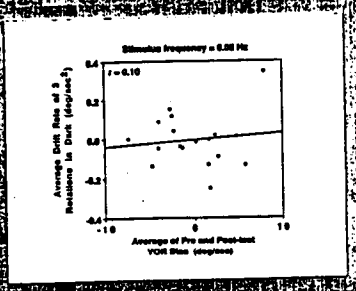
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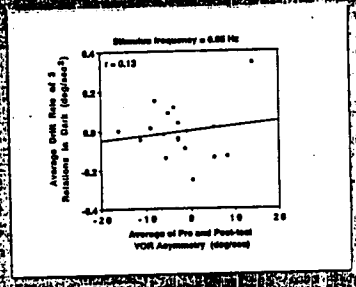
Most subjects showed a slow linear drift of velocity to one side, indicating a tendency to perceive velocity faster in one direction than the other.



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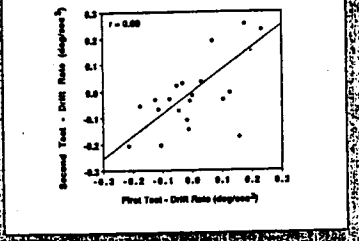
Correlations between drift rate and VOR bias and asymmetry were poor.

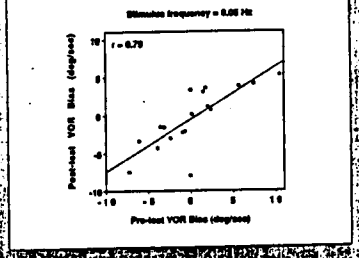




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Measurement errors could not account for these poor correlations since repeated tests of drift rate, VOR bias, and VOR asymmetry in the same subjects were highly correlated.





CONCLUSIONS

7

A simple imbalance in vestibular function between the two ears cannot account for both perceptual and VOR asymmetries. Central adaptive VOR mechanisms which balance and calibrate VOR function are well known. Perhaps a separate adaptive mechanism exists for motion perception. The measured perceptual and VOR biases may be related more to important function of these separate adaptive mechanisms, than to peripheral vestibular function.

AGE-RELATED CHANGES IN HUMAN VESTIBULO-OCULAR AND OCULOMOTOR REFLEXES

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Summary. The dynamic response properties of horizontal vestibulo-ocular reflex (VOR) and optokinetic reflex (OKR) were characterized in 216 human subjects ranging in age from 7 to 81 years. The object of this cross-sectional study was to determine the effects of aging on VOR and OKR reflex dynamics, and to identify the distributions of parameters which describe VOR and OKR responses to sinusoidal and pseudorandom stimuli in a putatively normal population. In general, VOR and OKR response parameters changed in a manner consistent with declining function with increasing age. For the VOR this was reflected in declining response amplitudes and slightly less compensatory phase of the response relative to head velocity. For the OKR the lag time of the response, which is probably associated with the time required for visual information processing, increased linearly with age at a rate of about 1 ms per year. Although age-related trends were clear, the magnitude of change of VOR properties were not large relative to the variability within the population. The age-related trends in VOR were also not consistent with the anatomic changes in the periphery reported by others which showed an increasing rate of peripheral hair cell and nerve fiber loss in subjects over 55 years. The poor correlation between physiological and anatomical data suggest that adaptive mechanisms in the central nervous system are important in maintaining the VOR.

Key Words: Vestibulo-ocular reflex - Optokinetic reflex - Eye movements - Aging - Humans

Introduction

A great deal is known about age-related performance declines in various visual perception tasks (Fozard et al. 1977). These tasks are usually studied in controlled settings in which oculomotor performance is assumed not to play an important role. However in natural settings it is obvious that changes in oculomotor performance with age could have a deleterious affect on many aspects of visual perception. In particular, VOR and OKR normally function together to provide clear vision by generating compensatory eye movements which minimize image motion on the surface of the retina during head movements. It is important to know how the VOR and OKR change with age since a degradation in these reflexes could impair the acquisition of visual information in older adults during active and passive head movements.

Both peripheral vestibular receptor dynamics and central nervous system processes influence the VOR. For rotations in the horizontal plane, the horizontal semicircular canals provide the primary peripheral signal of head rotation. Theoretical studies of semicircular canal biophysics (van Egmond et al. 1949) as well as recordings from primary canal afferents (Fernandez and Goldberg 1971) have shown that the canals effectively integrate the rotational acceleration so that the canals signal rotational head velocity at frequencies above about 0.25 Hz. If rotational velocity is considered to be the stimulus, then the dynamic response properties of the canal are those of a high-pass filter with a time constant of about 6 s (Fernandez and Goldberg 1971).

During rotations in the horizontal plane in the dark, compensatory (slow phase) eye movements are interspersed with fast components in the opposite direction resulting in a triangular shaped eye movement waveform (nystagmus). The fast components reposition the eyes toward the center of their range, or sometimes past their center position in a direction which anticipates the continued head movement. VOR response dynamics are typically analyzed by comparing the slow phase eye velocity to the stimulus velocity. VOR dynamics analyzed in this fashion show the high-pass filter characteristics similar to those of the semicircular canals themselves. However the time constant of the VOR is two to four times longer than those recorded from first order canal neurons. The brainstem function which causes this time constant lengthening is known as the velocity storage mechanism (Raphan et al. 1979; Cohen et al. 1981). The net result of this velocity storage is to extend the effective bandwidth of the VOR to frequencies below 0.25 Hz.

During horizontal head rotations in the light with earth fixed visual surrounds, the VOR and visual motion information through optokinetic and pursuit tracking systems combine to produce compensatory eye movements which facilitate clear vision by maintaining a fixed gaze direction. The combined VOR and visual tracking reflexes are effective over a bandwidth from DC to several Hertz (Benson and Barnes 1978; Larsby et al. 1984). The visual system's contribution to the generation of compensatory eye movements can be studied using rotations of a full field visual stimulus around a stationary subject. This stimulus evokes the OKR to produce eye movements which follow the stimulus. The response properties of the OKR have been determined primarily using responses to constant velocity rotations (Cohen et al. 1977). OKR response properties in humans and most other primates are characterized by a two component response. These two components are mediated by distinct

anatomical pathways (Waespe et al. 1983). The fast, or direct component is associated with the rapid increase in eye velocity following the initiation of visual field movements. The slow, or indirect component is associated with a slow increase in eye velocity following a sustained motion of the visual field. The slow component shares many of the central vestibular pathways, and possibly the velocity storage mechanism of the VOR (Buettner et al. 1978; Cohen et al. 1981). Experiments show that human OKR is dominated by the fast component (Cohen et al. 1981; Segal and Liben 1985).

Adaptability is another characteristic feature of the oculomotor reflexes. In particular the gain of the VOR can be adjusted in order to minimize motion on the retina. These adjustments occur over a period of minutes to days (Melvill Jones 1985; Lisberger 1988). Adaptation provides a mechanism for the calibration of the VOR. Adaptation also assists in the recovery of function following injury or diseases in the ear, brain, or oculomotor system. Deficits incurred during the normal aging process may also be ameliorated by these adaptive mechanisms.

Age-related changes in the peripheral vestibular organs include losses of hair cells (Rosenhall 1973), vestibular nerve fibers (Bergstrom 1973), and Scarpa's ganglion cells (Richter 1980). These studies showed an increased rate of loss of peripheral vestibular anatomical structures for subjects older than about 55 years. If reflex function depends directly on intact peripheral vestibular structures, then we might expect a decline in VOR function paralleling anatomical deterioration. Alternatively, if the central adaptive mechanisms remain intact in older subjects, then VOR function may remain relatively stable regardless of peripheral anatomical deterioration.

Literature on age-related changes in vestibular and oculomotor function is limited. Experiments on humans are usually performed on a small number of subjects, and these subjects are typically young adults. The exceptions to this occur in the clinical literature on caloric testing of the VOR (Bruner and Norris 1971; Mulch and Petermann 1979), some work on age-related changes in pursuit tracking ability (Sharpe and Sylvester 1978; Spooner et al. 1980) and VOR function (Wall et al. 1984; van der Laan and Oosterveld 1974), responses to constant velocity optokinetic stimuli (Magnusson and Pyykko 1986; Stefansson and Imoto 1986), and development of vestibular reflexes in infants and young children (Ornitz 1983; Shupert and Fuchs 1988). The caloric test results are rather ambiguous, and include both increased, decreased, and unchanged responses with increasing age. The results of caloric testing are therefore not consistent with the known age dependent anatomical changes in peripheral vestibular receptors and nerve fibers. However the increased number of falls which occur in the elderly population (Overstall 1978; Prudham and Evans 1981) suggest that there might be a connection between the occurrence of these falls and a generally declining function of vestibular and oculomotor reflexes associated with the control of orientation and equilibrium.

The present experiments were designed to characterize the dynamic response properties, and the variability of those responses of human horizontal VOR and OKR in a normal population. The population was selected to provide results related to the effects of the aging process on these reflexes, and to determine if physiological changes were consistent with anatomic changes which occur with age. Tests of postural motor control and sensorimotor

integration of visual, vestibular, and proprioceptive information used for posture control were also made in the same subjects and are reported in a companion paper (Peterka and Black 1989).

Methods (Note: Methods section should be in small print)

Vestibular and oculomotor reflexes were tested in 216 human subjects (90 male and 126 female) aged 7 to 81 years. Ages were approximately uniformly distributed over the entire range. Tests included 1) horizontal vestibulo-ocular reflex, 2) horizontal optokinetic reflex, 3) caloric irrigations, and 4) moving platform posturography. Various parameters which characterize the dynamic response properties of these reflexes were measured. Statistical methods were used to define the range of responses, and determine age-related changes in function.

Subjects were required to meet the following criteria:

1. normal age-corrected auditory pure tone responses
2. middle ear reflexes present bilaterally
3. normal middle ear impedance
4. no history of head blows of sufficient magnitude to cause loss of consciousness
5. no abnormal neurological findings
6. normal corrected vision
7. no history of ototoxic drug use
8. no history of dizziness or disequilibrium
9. moderate or absent use of alcohol
10. no use of psychotropic drugs

We did not reject subjects based on the results of any of the vestibular or oculomotor tests performed.

Rotation tests

Subjects sat in a chair mounted on an 108 N·m velocity servo-controlled motor (Contraves Goerz Corp, Model 824) which rotated them about an earth vertical axis. The subject was surrounded by a circular cloth cylinder 1.8 m in diameter. The cylinder acted as a projection screen for an optokinetic stimulus. A full field optokinetic stimulus was provided by a pin hole type projector mounted on a 6.8 N·m servo motor (Genisco Technology Corp Model 1100) attached to the ceiling directly above the subject's head. The projector produced randomly placed vertical stripes of light against a mostly dark background.

Subjects performed tests of VOR function with eyes closed in a darkened room. Horizontal and vertical eye movements were recorded by electrooculographic (EOG) techniques (bandwidth DC to 80Hz) using silver/silver chloride electrodes. Horizontal EOG was recorded using bitemporal electrodes, and vertical EOG was recorded by electrodes placed above and below one eye. Stimulus delivery and data collection were controlled by computer (DEC LSI 11/73). Chair tachometer signals as well as horizontal and vertical EOG were digitized and stored for later analysis. Digitizing rates were 200/s for the horizontal EOG and 50/s for vertical EOG and stimulus velocity.

Calibrations of the EOG were performed before and after each rotation test. Three red LED's mounted on the screen at 0 and +/- 10^{o} were successively illuminated. As the subject looked at the illuminated LED's, the computer recorded the voltage associated with each gaze angle. The EOG amplifier's gain was adjusted to provide adequate EOG signal amplitude relative to the digitizing resolution of the A/D converter (12 bits).

Rotational stimuli for VOR tests included both single frequency sinusoidal stimuli and a pseudorandom stimulus. Single frequency sinusoidal stimuli were delivered at 0.05, 0.2, and 0.8 Hz with peak velocities of 60^{o}/s. The duration of sine tests were 100 s (5 cycles) for 0.05 Hz, 45 s (9 cycles) for 0.2 Hz, 26.25 s (21 cycles) for 0.8 Hz. The first cycle in each data record was considered a transient response and was ignored in the data analysis.

The pseudorandom stimulus consisted of the summation of eight discrete sinusoidal frequencies. The frequency components were selected to minimize corruption of the results of the data analysis due to possible nonlinear responses of the VOR system (Victor and Shapley 1980). The eight frequencies were 0.0092, 0.021, 0.046, 0.095, 0.180, 0.388, 0.766, 1.535 Hz. The nominal amplitudes of these components were all 15.6^{o}/s except the highest frequency component which was 7.8^{o}/s. The highest instantaneous stimulus velocity was about 100^{o}/s. The duration of the stimulus was about 440 s. Data from the final 327.68 s of the trial were digitized and saved for later analysis. The first 115 s of data (about equal to one period length of the lowest frequency component of the stimulus) were considered a transient response and were not analyzed.

The OKR of each subject was tested by recording horizontal eye movements evoked by a projected visual stimulus rotated around the stationary subject. The optokinetic projector moved under the control of a pseudorandom stimulus consisting of seven sinusoids with frequencies 0.018, 0.043, 0.092, 0.189, 0.360, 0.775, and 1.532 Hz. The amplitudes of the components were nominally 7.8^{o}/s except the highest frequency component which was 3.9^{o}/s. The peak instantaneous velocity was about 40^{o}/s. The total stimulus duration was about 220 s. Transient responses were avoided by recording only the final 163.84 s of data. Complete OKR data were not obtained on all subjects since the stimulus induced motion sickness symptoms in some subjects, requiring the early termination of the test.

Subjects were given verbal tasks throughout the VOR and OKR rotation tests to maintain a constant level of alertness. The tasks consisted of alphabetically naming names, places, foods, etc.

Rotation Test Data Analysis

Eye position data were differentiated to calculate eye velocity. Fast phases of the nystagmus were identified using a method similar to Barnes (1982). Curve fits to the remaining slow phase eye velocity data allowed the estimation of VOR and OKR response parameters. For sinusoidal stimuli, curve fits were made to each period of the response. Stimulus periods which contained corrupted data were rejected before the final averaging of response parameter values from the remaining periods.

The curve fits to sinusoidal responses were of the form:

$$r(t) = B_r + A_r \sin(2\pi f + P_r) \qquad (1)$$

where B_r is bias in deg/s, A_r is response amplitude in deg/s, P_r is response phase in degrees, and f is the stimulus frequency. The recorded chair velocity data were separately analyzed to calculate stimulus velocity amplitude, A_s, and phase, P_s. The VOR gain of the reflex is defined as the ratio A_r/A_s, and the phase of the reflex as P_r-P_s. Since the VOR is a compensatory reflex, the values of P_r-P_s was close to -180^0. For the convenience of working with smaller numbers, a value of 180^0 was added to P_r-P_s for the VOR test. This is equivalent to inverting the horizontal eye position data. Since the OKR is a following reflex, the value of P_r-P_s is close to 0^0 for low frequency stimuli. Therefore no offset factor was applied to the OKR phase data.

In order to identify and quantify nonlinear responses to sinusoidal stimuli, the horizontal eye velocity data was shifted in time by an amount determined by the calculated phase of each period of the response. The time shift was in a direction which brought the response into phase with the stimulus. Slow phase eye velocity was then plotted against stimulus velocity to yield a scatter of points which generally lie along a negatively sloping line. An example is shown in Figure 1. The negative slope for the VOR reflects the fact that slow phase eye velocity is in the opposite direction to stimulus velocity. The slope of the line is equal to VOR gain.

If the VOR were a linear system, the slope of the data would be the same for eye movements to the right and left. However experience with abnormal subjects has shown that the slopes are not always equal for chair rotations in opposite directions. This type of nonlinearity was quantified by separately calculating the slopes of the eye velocity versus stimulus velocity data for chair rotations to the right and left. The slopes were calculated by a least squared error fit of a two segment line to the data. One line segment was for positive and the other for negative stimulus velocities. The two line segments were constrained to intersect one another at zero stimulus velocity.

The two-part linear curve fit yields three parameters: the reflex gain for slow phase eye movements to the right, G_R, the gain for slow phase eye movements to the left, G_L, and response offset defined as the eye velocity at zero stimulus velocity. A measure of response asymmetry was calculated according to the formula $100*(G_R-G_L)/(G_R+G_L)$. A zero percent asymmetry is consistent with a linear system response where gain is independent of the stimulus direction.

The analysis of eye movement data from pseudorandom stimuli followed the same general method used in single sine analysis. The Fourier analysis of the slow phase eye movement data provided estimates of the response parameters given by the following equation:

$$r(t) = B + \sum_{i=1}^{N} A_i \sin(2\pi f_i + P_i) \qquad (2)$$

were B is bias or average slow phase velocity, with units of deg/s, N is the number of sinusoidal components in the pseudorandom stimulus, A_i is the response amplitude at the i^{th} frequency f_i, and P_i is the response phase at the i^{th} frequency. A Fourier analysis of the stimulus velocity was performed to calculate the amplitudes and phases of the stimulus waveform. The reflex gains and phases at the N stimulus frequencies were computed as the ratio of response amplitude to stimulus amplitude, and the difference between response phase and stimulus phase, respectively. As with single frequency sinusoidal stimuli, a value of 180^o is added to the calculated phases for tests of the VOR reflex.

The gain and phase values of the VOR reflex were fitted with a transfer function equation (Seidel 1975) of the following form:

$$H_{vor}(s) = \frac{K_V T_V s}{T_V s+1} \qquad (3)$$

where T_V is an estimate of the VOR time constant (units of seconds), K_V is the VOR gain constant, and s is the Laplace transform variable.

OKR gain and phase data for all subjects were well fit by a three parameter transfer function of the form:

$$H_{okr}(s) = \frac{K_o \exp(-T_d s)}{T_o s+1} \qquad (4)$$

where T_o is a time constant with units of seconds, K_o is the OKR gain constant relating slow phase eye velocity to stimulus velocity, and T_d is a time delay parameter with units of seconds describing the lag between visual field movement and eye movement. The $T_o s+1$ factor represents a lowpass filter which accounts for the declining gain with increasing frequency observed in some subjects. Larger values of T_o are consistent with gain declines beginning at lower frequencies. A value of zero for T_o (i.e. the transfer function reduces to $H_{okr}(s) = K_o \exp(-T_d s)$) accounts for subjects whose gain did not decline with increasing frequency. T_o is not the time constant associated with velocity storage and optokinetic afternystagmus.

Caloric Tests

Four irrigations of the external ear canals were made using a Brookler-Grams closed loop caloric irrigator. Subjects were in a supine position with head elevated about 30^o above horizontal to assure maximal stimulation of the horizontal semicircular canals. The caloric test was not performed on subjects under 12 years, and complete data were not obtained on other subjects since some became nauseated or simply chose not to continue the irrigations because of discomfort. Each ear was alternately irrigated for 45 s at 30 and 44^oC. Horizontal and vertical eye movements were recorded with EOG techniques identical to those described for rotation tests. Eye movements were recorded during and after each irrigation for a total of 3 minutes. Horizontal eye movements were analyzed to calculate peak slow phase

eye velocity. A comparison of peak velocities for the two ears provided an indication of the balance of sensitivity to thermal stimulation between the two ears. This comparison was quantified by the unilateral weakness measure defined by:

$$UW = \frac{(RW + RC) - (LW + LC)}{RW + RC + LW + LC} \times 100 \qquad (5)$$

where RW, RC, LW, LC are the absolute values of peak slow phase eye velocities recorded during right warm, right cold, left warm, and left cold irrigations, respectively (Barber and Stockwell 1980). Subjects were tasked throughout caloric testing to maintain alertness.

Curve Fits to Aging Data

In order to visualize trends in various response parameters as a function of age, a robust locally weighted regression analysis (lowess fit) was performed to obtain smoothed scatterplots (Cleveland 1979 and 1985). A lowess fit parameter of f=0.5 and iteration parameter of 2 were used on all data sets.

Data Quality

The overall quality of each rotation and caloric test for each subject was subjectively given a rating of good, fair, or poor. Only good and fair quality data are included in the data summaries in the results section. Quality judgments were based on the standard deviation of response parameters (such as gain, phase, and bias from rotation tests), on the consistency of the responses throughout the duration of the stimulus, and on the accuracy of the eye movement analysis in the separation of slow and fast phases of nystagmus. The actual values of response parameters were not used in judgment of data quality. The test results from about 4 percent of subjects were rated poor for each test. Poor quality data for one subject on a given test was not used to disqualify other data from the same subject on other tests.

Results

The subjects showed a wide range of responses on most measures of vestibulo-ocular and oculomotor function. Age-related changes were identified in almost all rotation test response measures, but the magnitude of these changes was not large relative to the variability of the data. Most changes indicated a decline in function. In contrast, no obvious or consistent changes as a function of age were found in caloric test responses. There were no significant differences in reflexes between males and females.

VOR Responses

Typical VOR test results from a sinusoidal rotational stimulus are shown in Figure 1. Box graph distributions of gain, phase, bias, offset, and asymmetry are shown for the entire population at all test frequencies in Figures 2. Table 1 summarizes the statistics of these distributions. The small differences in N's are due to data eliminated because of poor quality. These distributions are reasonably symmetrical about their means. Gain

increased with increasing frequency and had lower variance at 0.8 Hz as compared to 0.2 and 0.05 Hz. The phase variance also decreased with increasing frequency. The higher variance of the 0.05 Hz phase reflects the variability of the VOR time constant in the population. Since phases at higher frequencies are not affected by the VOR time constant their distributions had lower variances. The variance of the offset distributions are all somewhat less than the variances of the bias distributions. This is consistent with the fact that both nonlinear responses (asymmetric gains) and nonzero average slow eye velocity contribute to the bias measure. That is, a portion of the bias measure is accounted for by the presence of asymmetrical gains. The remaining portion, which is the response offset, is therefore smaller than the bias.

A sample of a typical response to a pseudorandom VOR stimulus is shown in Figure 3. The pseudorandom stimulus evokes a complex eye movement pattern (Figure 3C). However separation of slow and fast components, and calculation of slow phase eye velocity reveals the underlying compensatory motion (Figure 3B). A spectral analysis of slow eye velocity and the recorded stimulus velocity provides measures of response gain and phase as a function of stimulus frequency. Examples of gain and phase data from three subjects are shown in Figure 4. Typically the gain is lower at the lowest test frequency and increases with increasing frequency. In some subjects the gain monotonically increases over the frequency range tested and in others it appears to reach an asymptote. The solid lines through the data points represent curve fits of a two parameter transfer function (equation 3) to the data of each subject.

The pattern of VOR gain and phase data of most subjects were similar in form to those in Figure 4A, and were well characterized by the two parameter transfer function model (equation 3). However there were deviations from this pattern which are exemplified by the data from two other subjects in Figures 4B and C. The low frequency data in 4B was fit well by the two parameter model but the higher frequency data showed increasing phase leads with increasing frequency. The phases of VOR responses to 0.05, 0.2, and 0.8 Hz sinusoidal rotations were 10.7^{o}, 2.0^{o}, and 6.9^{o}, respectively, for this subject and therefore confirmed the general pattern. A more accurate curve fit to this data would require a higher frequency lead term in the transfer function. A transfer function of this form would be similar to the one used to describe the dynamic responses of phasic canal afferents in the squirrel monkey (Fernandez and Goldberg 1971).

The VOR phase data in Figure 4C was fairly flat and greater than zero across all test frequencies. The phase of responses to sinusoidal stimuli were 3.7^{o}, -2.8^{o}, and 2.1^{o} at 0.05, 0.2, and 0.8 Hz, again confirming the general pattern but with less phase lead than the pseudorandom data. The curve fit identified a long VOR time constant of 44.9 s. However the two parameter model does not describe either the low or high frequency phase data well. A transfer function fit with a s^{k} Laplace operator is better able to describe this type of data (Anastasio and Correia 1988).

Pseudorandom stimulus test results are displayed in Figure 5 as box graphs of VOR gain and phase data as a function of stimulus frequency. As with single frequency sinusoidal results, the variance of gain data were similar across all frequencies. The variance of phases data was larger at low frequencies

than at high probably reflecting the variance of the VOR time constant among individuals. The variance of the phase data at 1.535 Hz was larger than the phase data at adjacent lower frequencies. This probably resulted from the fact that the stimulus amplitude of the highest frequency component was half that of the lower frequencies resulting in a lower signal-to-noise ratio at the 1.535 Hz test frequency.

The distribution of the VOR gain constants and time constants for the pseudorandom stimulus are shown in Figure 6 and summarized in Table 2. The gain constant distribution was symmetrical with an average value of 0.72. The VOR time constant distribution was skewed toward longer time constants. Mean value was 24.4 s and median value was 23.0 s. Only two subjects had time constants below 10 s. One of these subjects (time constant = 8.2 s) had a partial unilateral loss of vestibular function as judged by caloric testing. A short time constant is consistent with a unilateral loss of vestibular function as reported in animal models and humans (Wolfe and Kos 1977; Paige 1983; Honrubia et al. 1984; Peterka and Black 1987). The other subject (time constant = 7.0 s) had normal caloric test results. Therefore the abnormally short time constant for this subject remains an unexplained anomaly.

Comparison of VOR Measured by Single Frequency and Pseudorandom Stimuli

If the VOR were a linear system, then gain and phase data obtained from single frequency sinusoidal and pseudorandom stimulation should be identical within the random variability introduced by measurement errors. Statistical comparisons were made between the single frequency and pseudorandom gains and phases and are shown in Table 3. Single sine and pseudorandom results were not significantly different at the lowest frequency (0.05 Hz). Small but consistent differences were evident at higher frequencies. In particular, the single frequency gain was higher at 0.8 Hz than the pseudorandom derived gain and the pseudorandom phase values at 0.2 and 0.8 Hz were phase advanced by about 3^{o} compared to single frequency sine results. The improved phase response from sine tests may represent a small predictive effect. However this effect did not apparently carry over to the 0.05 Hz data.

The gain value from the 0.8 Hz test was higher than the pseudorandom test result. This might also be due to a predictive effect. However, in this case it seems likely that the data analysis methods could have contributed to this higher value. During the analysis of the single frequency sine tests, the experimenter had the ability to reject data from stimulus cycles which were obviously corrupted. These corrupted data cycles could easily be identified based on gain, phase, and/or bias values which deviated greatly from the values for other cycles. There were several causes for poor data cycles including transient increased EMG interference as a subject squinted, excessive blinking, looking up or down, inattentiveness to tasking, and failure of the fast phase eye movement detection algorithm. With experience, it became a simple task to detect and correct these problems by rejecting the affected cycles. The net effect usually increased the average gain measure. Eye movement recordings were also transiently corrupted during pseudorandom testing. However we did not have a means of correcting or eliminating these problems and they were therefore averaged into the final result. Therefore the rejection of corrupted portions of single sine results, but not pseudorandom results, could account for the higher gains measured during sinusoidal rotations.

VOR Responses to Caloric Stimuli

Caloric test results are generally in agreement with those of others (Barber and Stockwell 1980) who report normal ranges of unilateral weakness of about 15 to 25%. Our results are consistent with a 25% upper limit of normal since 95% of our subjects had unilateral weakness measures below this value. The distribution of peak slow phase eye velocity had a mean of 17.0°/s (+/- 9.0 s.d., range 4.5 to 63.2) and was skewed toward larger values.

OKR Responses

Typical OKR test results from pseudorandom stimulation for two subjects are shown in Figure 7. Response gain was less than unity in all subjects. The gains of most subjects were approximately flat across the bandwidth of frequencies tested (0.02 to 1.5 Hz) as in Figure 7A. Phase was near 0° at the lowest frequencies and showed monotonic increasing phase lags as frequency increased. Since perfect tracking of the visual stimulus is represented by unity gain and zero phase at all frequencies, subjects demonstrated very imperfect tracking in terms of both amplitude (gain) and timing (phase). OKR response bias was near zero for all subjects. The major variation on the typical OKR result was the presence of declining gain with increasing frequency in some subjects. Figure 7B shows the OKR transfer function data from one such subject.

The means, standard deviations, and ranges of OKR gain constant, time constant, time delay, and bias are given in Table 4. The distribution of these parameters are shown in the histograms in Figure 8. Both the gain constant and time delay have approximately symmetric distributions. In contrast, the OKR time constant has a highly skewed and possibly bimodal distribution with about 40 percent of the values near zero. OKR time constants near zero reflect the fact that OKR gains for these subjects were approximately constant over the frequency range tested.

The OKR pseudorandom stimulus was quite provocative in the initiation of motion sickness symptoms. Twenty subjects requested the termination of testing as a result of the onset of motion sickness symptoms. Approximately an equal number experienced motion sickness symptoms but were able to complete the 220 s duration OKR stimulus. It was not possible to calculate OKR gains and phases from incomplete trials using our current analysis methods. Therefore it was not possible to test the hypothesis that abnormal OKR responses were related to motion sickness sensitivity in these highly susceptible subjects. However, OKR gains and phases from subjects who reported the onset of motion sickness symptoms but were able to complete the test did not show any obvious differences compared with subjects who did not report symptoms. Also comparisons of VOR rotation test results of OKR motion sickness susceptible subjects with nonsusceptible subjects did not reveal any differences between these populations.

Age-Related Changes in VOR, OKR, and Caloric Responses

Several VOR and OKR response parameters changed with age (Figures 9-12). However all rotation test measures of VOR response symmetry (the absolute values of VOR bias, offset, and asymmetry) did not. The caloric test measure of response symmetry (unilateral weakness) was constant until age 55 years, and then showed an upward trend (Figure 12B).

Many of the age-related changes showed roughly linear trends. Linear regression slope, intercept, and correlation coefficients are summarized in Table 5. In particular, all single sine gains (Figure 9) decreased with increasing age. The gain trend was more consistent at 0.05 Hz than at 0.2 and 0.8 Hz. Single sine response phases increased with increasing age at all frequencies tested, although the effect was more pronounced at 0.2 and 0.8 Hz than at 0.05 Hz. Both VOR time constant derived from pseudorandom results (Figure 10B), and OKR gain constant (Figure 11A) increased slightly in subjects up to about 30 years, and then decreased with increasing age. The OKR time delay parameter increased with increasing age. The OKR time delay showed the clearest age-related trend ($r = 0.53$ and slope = 1.2 ms/year) of all VOR and OKR parameters.

The age-related change in the OKR time constant was clearly not linear. Data in Figure 11B show that a large number of subjects between about 20 and 60 years had OKR time constants close to zero, indicating that their OKR gain was constant across frequency. In contrast, there were very few subjects under 20 years and proportionally fewer of the subjects over 60 which had zero OKR time constants, indicating that on average their OKR gain declined with increasing frequency. The lowess curve fit indicates that age-related trends were minimal for subjects between 20 and 60 years. Subjects under 20 years showed an age-related decline in their OKR time constant with increasing age. Subjects over 60 years showed an age-related increase in their OKR time constant with increasing age.

Age-related effects on caloric test results were ambiguous. A linear regression curve fit to the data in Figure 12A showed an average decrease in peak slow phase eye velocity with increasing age. The linear regression had an associated correlation coefficient of -0.15 which is significantly different from zero ($P<0.05$). However the lowess fit shown in Figure 12A indicated that a linear regression was probably not an appropriate description of the data. Peak slow phase velocity decreased for subjects up to about 40 years, and then increased at a low rate for older subjects.

Figure 12B plots the absolute value of the unilateral weakness measure against age. The lowess fit shows essentially no change over the first 6 age decades, and a slight increase in older subjects. Due to the large variance in the data, a much larger sample would be required to determine if the small increase in older subjects was significant.

Discussion

We were able to clearly identify age effects on vestibular and oculomotor reflexes. The direction of change of some reflex parameters were expected, such as declining VOR gains, declining OKR gains, and increased time delays in the OKR with increasing age. Other age-related changes were not expected. These include increased VOR phase leads with increasing age, the fact that VOR function determined using caloric testing did not show the same trend as VOR function measured using rotation tests, and decreased high frequency OKR gain of the youngest and oldest subjects as compared to middle aged subjects.

VOR Changes with Age

Age-related changes in the VOR do not appear to reflect age-related peripheral vestibular anatomical changes. Figure 13 shows the lowess curve fit to 0.8 Hz VOR gain versus age plotted along with curve fits to data on human crista hair cell counts (Rosenhall 1973), vestibular nerve fibers (Bergstrom 1973), and Scarpa's ganglion cells (Richter 1980) as a function of age. The ordinate scales are linear and were normalized to their values at a subject age of 30 years. For ages up to about 50 years there is a gradual decline in both VOR gain and the various measures of peripheral vestibular anatomic components. For the VOR gain this gradual decline continues at about the same rate into the higher age decades. However the rate of decline of all anatomic measures greatly increases after about age 60, resulting in a divergence between the anatomical and physiological data. The net effect is that the VOR functions better in subjects older than 60 years than would be predicted based on changes in peripheral vestibular anatomy.

Because the subjects of this study were volunteers, it could be argued that the sample of older subjects would be biased in favor of exceptionally healthy elderly individuals who do not reflect the physiological function of a randomly selected population. This seems unlikely for several reasons. The auditory pure tone threshold functions of our subjects were consistent with the expected age-related changes (Rowland 1980). Additionally extended frequency audiometry (8-20 KHz) was performed on most subjects and showed consistent monotonically declining function with increasing age. The OKR time delay also showed monotonic increases with age. Finally, posture test results (Peterka and Black 1989) in the same subjects showed clear age effects which appeared more closely related to the time course of peripheral vestibular anatomical changes. It seems unlikely that the peripheral vestibular system of these subjects would escape distributed aging processes when other systems did not.

The increases in VOR phase leads at higher frequencies with increasing age were not anticipated. On the surface they would seem to represent a degradation of function since increased phase leads take the system response away from the goal of perfect compensatory eye movements (unity gain and zero phase). Perhaps the phase advance is an artifact of an adaptation which improves overall VOR function. For example, studies of peripheral semicircular canal function in the squirrel monkey have shown that higher gain peripheral nerve fibers have dynamic properties which include phase advances at higher frequencies (Fernandez and Goldberg 1971). Phase advances indicate a sensitivity to the velocity of cupula deflection in addition to the cupula position (which is assumed in the classical description of canal dynamics). In contrast, lower gain canal fibers show cupula position sensitivity and therefore, due to the integrating accelerometer characteristics of canal biophysics, the nerve responses are in phase with head velocity at higher frequencies of rotational movements.

On the basis of our results, we might postulate that in young people, low gain tonic canal fibers provide the major contribution to the VOR. As the subject ages and there is a gradual loss of peripheral canal input due to cell death, adaptive mechanisms in the central nervous system may be able to selectively increase the contribution of high gain canal nerve input. The sum effect would be to maintain the gain of the VOR at a reasonable level

allowing for the generation of adequate compensatory eye movements. However this mechanism of gain enhancement would be accompanied by the possibly undesirable phase leads associated with the dynamics of the high gain canal fibers. One could further hypothesize that a trade-off is occurring between maintaining the desirable feature of high VOR gain and the undesirable feature of phase advances in a direction away from perfect eye movement compensation. Simply stated, there appears to be a trade-off in favor of maintaining response amplitude at the expense of timing with increasing age.

This hypothesis may be consistent with the multichannel model of the VOR developed by Miles et al. (1985) to explain the dynamic properties of VOR adaptation. However, assuming that human and monkey VOR adaptation occurs by similar mechanisms, there are other studies which are not consistent with this hypothesis. Minor and Goldberg (1986) have shown that phasic canal fibers do not appear to contribute at all to the VOR of the squirrel monkey. If phasic fibers do not contribute to the VOR, then they cannot participate in alterations in VOR dynamics. One might argue that these phasic fibers only contribute to the VOR when they are needed for the adaptive enhancement of the reflex. However this would be inconsistent with other results which suggest that it is an enhancement of the contribution of the tonic fibers which mediates adaptive increases in VOR gain (Lisberger and Pavelko 1986; Lisberger 1988).

Finally, a hypothesis calling for an increased phasic fiber contribution to the VOR of older subjects may also be inconsistent with anatomical aging results which showed relatively greater hair cell loss on the crest of the crista (Rosenhall 1973), and the greatest losses of the thick fibers innervating the canal cristas (Bergstrom 1973). Since the higher gain afferent fibers, at least in the guinea pig and the squirrel monkey, tend to be larger in diameter and to originate from the crest of the crista (Goldberg and Fernandez 1977; Fernandez et al. 1988; Baird et al. 1988), the selective loss of these cells with increasing age would obviously preclude their participation in VOR gain enhancement.

Current understanding of the mechanisms of VOR adaptation and of anatomical changes in peripheral vestibular receptors does not allow an easy fit to our VOR data. VOR adaptation has generally been studied in young or young adult animals. Perhaps the aging process also effects the functionality of the central neural networks involved in the adaptive process. An aging adaptive neural network could contribute its own dynamic component to the VOR which differs from those observed in younger animals.

OKR Changes with Age

The rate at which the OKR time delay increased with age was quite large, and is similar to the changes found in pursuit latency with increasing age (Sharpe and Sylvester 1978; Spooner et al. 1980). If this increased time delay is representative of general changes in the speed of visual system motion processing associated with visuo-motor tasks, this could affect tasks, such as posture control, which use vision for feedback control. Longer feedback time delays generally contribute to decreased stability and poorer overall performance.

However the interpretation of age-related changes in OKR time delay is complicated by the fact that the OKR time constant and time delay parameters are probably not statistically independent. This is because the lag term $T_o s+1$ in equation (4) which accounts for the declining gain at higher stimulus frequencies also accounts for some of the phase lag. The larger the OKR time constant, the more phase accounted for by the lag term, and therefore the smaller the value of the OKR time delay parameter required to explain the remaining phase lag. Since the youngest subjects had the largest OKR time constants, this would tend to bias their time delay parameters toward lower values. The oldest subjects also tended to have larger OKR time constants which should also bias their time delays toward lower values. However Figure 11C shows that, despite this possible bias, older subjects had the largest time delays in the population. This indicates that the time delay associated with visual processing of motion information must truly be increasing with age. However the time course of time delay increase in Figure 11C may be distorted by the interaction with the OKR time constant parameter.

When individuals' OKR data were fit with a transfer function model of the form $K_o \exp(-T_d s)$, which did not include the lag term in equation (4), a different pattern of change of T_d with age emerged. On average this new T_d was about 50 ms longer than the T_d from equation (4) fits. Subjects younger than 16 years had time delays averaging about 240 ms. Subjects aged 20 to 25 years had the shortest time delays of the entire population; averaging about 210 ms. T_d increased with increasing age in subjects older than 25 years. The rate of increase was about 0.8 ms/year for subjects up to about age 50 years, and then about 1.6 ms/year for subjects over 50 years.

The changes in OKR time constant with age were not expected. Both younger (<15 years) and older (>65 years) subjects were relatively less responsive to the higher frequency components of the stimulus. The lower OKR responsiveness at higher frequencies could have functional consequences, particularly for older subjects. While it is generally appreciated that visual tracking reflexes improve visual-vestibular generated compensatory eye movements during low frequency head movements, visual motion information is apparently used to improve the dynamics of compensatory eye movements at higher stimulus frequencies associated with natural head movements (Peterka et al. 1987). This would be particularly important for individuals who had VOR phase leads at higher frequencies (Figure 4B). Since older individuals had larger VOR phase leads on average than younger subjects, we might expect that the older subjects would need more help from their visual tracking reflexes to correct the imperfect VOR dynamics. However the sensitivity to optokinetic motion at higher frequencies declined in many older subjects making it less likely that visual tracking reflexes could correct for imperfect VOR dynamics.

Clinical Significance

The presence of age-related changes in oculomotor reflex function has implications for the assessment of normal function. Part of the variability of VOR response parameters is caused by this age effect. The square of the correlation coefficient gives an estimate of the proportion of variance related to changes with age. The VOR gain versus age measures had correlation coefficients between 0.3 and 0.4. Therefore approximately 10 to

15% of the variance of gain data is accounted for by the aging affect. The largest correlation coefficient was 0.53 for the OKR time delay indicating that 28% of the variance was due to the effect of aging. Normal scales of vestibular and oculomotor function should account for these age effects.

Since the majority of the observed response variability is independent of age, it is clear that the functional characteristics vary widely within any given age group in a putatively normal population. To the extent that the aging affects are deleterious and that our reflex measures accurately characterize the general decline in function, a significant proportion of subjects within any age group look "older" than their chronological ages and are therefore less functional with regards to their orientation control abilities. One could hypothesize that these subjects would be more susceptible to the development of balance and orientation control problems as they age since we could expect their function to further decline with age. Perhaps there is some threshold beyond which the brain's adaptive mechanisms are not able to compensate for the declining function. After this point is reached, subjects may develop dizziness and equilibrium control complaints, or perhaps individuals will restrict their activities so as to avoid situations which stress their remaining capabilities. A longitudinal rather than a cross-sectional study would be required to test this hypothesis.

The aging process does not have a uniform effect on the reflex components involved in gaze stabilization during head movement. The optokinetic system presents the most complex picture with different parameters of the reflex showing different patterns of change with age. The VOR changed relatively little with age. This relative constancy was probably conferred by central adaptive mechanisms correcting for declining peripheral vestibular function. However central adaptive mechanisms cannot sustain VOR function indefinitely in the face of an increasing loss of peripheral receptors and neural substrate. It will be important to extend the age limit of our study to the eighth and ninth decades, and to explore larger amplitude and higher frequency stimuli which more nearly resemble natural head motion in order to find the point at which physiological function begins to follow the anatomical decline. This point will define the effective functional reserve of the central adaptive mechanisms.

Acknowledgment. We wish to thank Christopher Newell, Patrick Shea, and Martha Benolken for their assistance. This research was supported by NASA research grants NCC9-8 and NAG 9-117.

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Table 1. VOR response parameters for single sine stimuli. VOR phase is in degrees, bias in deg/s, offset in deg/s, and asymmetry in percent. Seven percentile values on the distributions of the parameters are given.

Frequency = 0.05 Hz, N = 208

	Gain	Phase	Bias	Offset	Asymmetry
Mean	0.68	10.5	-0.44	-0.35	-0.9
S.D.	0.15	4.85	3.56	2.90	7.5
2.5%tile	0.39	0.94	-8.21	-6.11	-18.8
5%	0.44	2.47	-6.34	-4.84	-15.1
25%	0.58	7.54	-2.55	-2.03	-5.7
50%	0.67	10.27	-0.36	-0.32	-0.5
75%	0.78	13.41	1.89	1.46	4.0
95%	0.96	18.10	5.00	3.84	10.8
97.5%	1.02	19.19	6.73	6.46	13.8

Frequency = 0.2 Hz, N = 208

Mean	0.75	1.62	-0.62	-0.35	-1.5
S.D.	0.16	3.17	2.91	2.32	6.6
2.5%	0.40	-4.27	-6.58	-5.00	-14.4
5%	0.51	-3.58	-5.70	-3.98	-11.7
25%	0.65	-0.36	-2.46	-1.79	-5.9
50%	0.75	1.96	-0.65	-0.33	-1.5
75%	0.85	3.97	1.32	1.09	3.2
95%	0.99	5.95	4.32	3.47	9.7
97.5%	1.02	6.39	4.88	4.49	10.4

Frequency = 0.8 Hz, N = 204

Mean	0.84	0.79	-0.28	0.04	-1.4
S.D.	0.13	2.59	2.76	3.13	5.9
2.5%	0.59	-3.95	-6.27	-6.78	-14.6
5%	0.62	-3.07	-5.30	-5.20	-11.9
25%	0.76	-0.59	-1.98	-1.62	-4.0
50%	0.84	0.74	-0.21	-0.10	-1.1
75%	0.93	2.05	1.52	1.86	1.6
95%	1.06	5.38	3.91	5.45	8.5
97.5%	1.07	6.99	5.14	6.50	10.3

Table 2. VOR response parameters for pseudorandom stimulus. VOR time constant is in seconds and bias is in deg/s. Seven percentile values on the distrubutions of the parameters are listed. N = 207 subjects.

	Gain Constant	Time Constant	Bias
Mean	0.720	24.50	-0.09
S.D.	0.156	8.56	2.14
2.5%	0.423	13.15	-4.96
5%	0.482	14.22	-3.75
25%	0.611	18.41	-1.34
50%	0.727	23.11	-0.02
75%	0.814	28.11	1.32
95%	0.974	43.59	3.18
97.5%	1.015	47.38	3.70

Table 3. Comparison of single sine and pseudorandom gain and phase results. Positive differences indicate that average single sine parameter value was larger than average pseudorandom parameter value. A * indicates that the difference was significant at $P<0.05$ using a paired variable Student t test comparison. The average differences listed are corrected for the difference in test frequencies between the single sine and pseudorandom stimuli. Gain and phase corrections were based on the VOR transfer function in equation (3) with average time constant of 24.5 s and gain constant of 0.72. N's are smaller than those in Tables 1 and 2 since comparisons were not made if either test had poor quality data.

Parameter	Single Sine	Pseudorandom	Average Difference	N	Significance
Gain	0.05 Hz	0.046 Hz	-0.0106	199	
	0.2 Hz	0.180 Hz	0.0185	199	*
	0.8 Hz	0.766 Hz	0.0951	195	*
Phase	0.05 Hz	0.046 Hz	-0.34	199	
	0.2 Hz	0.180 Hz	-3.20	199	*
	0.8 Hz	0.766 Hz	-3.44	195	*

Table 4. OKR response parameters for pseudorandom stimulus. OKR time constant and time delay are in seconds, and bias is in deg/s. Seven percentile values on the distributions of the various parameters are listed. N = 179 subjects.

	Gain Constant	Time Constant	Time Delay	Bias
Mean	0.651	0.080	0.180	-0.12
S.D.	0.116	0.080	0.043	0.84
2.5%	0.403	0.002	0.099	-1.67
5%	0.469	0.003	0.114	-1.52
25%	0.586	0.008	0.147	-0.68
50%	0.664	0.063	0.187	-0.10
75%	0.721	0.115	0.216	0.32
95%	0.807	0.226	0.248	1.13
97.5%	0.867	0.252	0.253	1.36

Table 5. Age effects on VOR and OKR response parameters. All parameter values which showed significant or nearly significant linear trends with age are listed. Correlation coefficients which were significantly different from zero ($P<0.05$) are marked with a *.

Parameter	Slope (change/year)	Intercept at 0 years	Correlation Coefficient	N
Gain - 0.05 Hz	-0.00295	0.795	-0.39*	208
- 0.2 Hz	-0.00261	0.849	-0.34*	208
- 0.8 Hz	-0.00215	0.926	-0.33*	204
Phase - 0.05 Hz	0.0289	9.32	0.12	208
- 0.2 Hz	0.0415	-0.03	0.27*	208
- 0.8 Hz	0.0498	-1.22	0.39*	204
VOR Gain Constant	-0.00188	0.794	-0.24*	207
VOR Time Constant	-0.0623 s	27.0 s	-0.15*	207
OKR Gain Constant	-0.00153	0.712	-0.26*	179
OKR Time Delay	0.00115 s	0.134 s	0.53*	179

Figure Legends

Fig. 1. Example of data from sinusoidal stimulus VOR rotation test. Upper trace shows slow phase eye velocity response to a 0.05 Hz, 60^{o}/s peak velocity sinusoidal rotational stimulus. Solid curve through the data is the curve fit to each cycle. Response gain, phase, and bias are obtained from these curve fits. Lower left traces show horizontal eye movements evoked by one period of the rotational stimulus. Solid vertical bars under the horizontal EOG trace show the location of fast phase portions of the nystagmus identified during the analysis. Lower right plot shows slow phase eye velocity plotted against stimulus velocity. The two part linear fit is used to measure response symmetry of VOR gain.

Fig. 2. Distributions of VOR response parameters at three different test frequencies. Data are plotted as box graphs. The line through the center box of each box graph shows the median value for the population. The central box encompasses 50 percent of the population, and the error bars encompass 95 percent of the population. Individual values below the 2.5 percentile and above the 97.5 percentile of the distributions are plotted separately. Bias and offset parameters are plotted as pairs of box graphs, with offset on the right of each pair.

Fig. 3. A 30 s sample of eye movements evoked by pseudorandom stimulation of the VOR. (A) shows the subject's rotational velocity, (B) slow phase eye velocity, (C) horizontal EOG, and (D) vertical EOG. Vertical bars between (B) and (C) show the locations of fast phases of the nystagmus detected by the computer analysis of the data.

Fig. 4. Examples of VOR gain and phase data from three subjects derived from responses to a pseudorandom rotation. Solid line through the data show the transfer function curve fit (equation 3). Gain and frequency scales are logarithmic.

Fig. 5. Box graphs showing the distributions of VOR gains and phases obtained from pseudorandom rotations as a function of frequency for 207 subjects. See Figure 2 legend for box graph description.

Fig. 6. Histograms showing the distributions of VOR gain and time constants estimated from curve fits to gain and phase data obtained from pseudorandom rotation tests.

Fig. 7. Examples of OKR gain and phase data from two individuals derived from responses to pseudorandom optokinetic stimulation. Solid lines show transfer function curve fits to data. Equations of the curve fits are inset. Gain and frequency scales are logarithmic.

Fig. 8. Population distributions of OKR gain constant, time constant, and time delay parameters derived from transfer function curve fits.

Fig. 9. VOR gain and phase as a function of subject age. Data were obtained from sinusoidal rotational stimulation at three different test frequencies. Solid curves are lowess fits.

Fig. 10. VOR gain constant (A) and time constant (B) parameters as a function of subject age. Parameter values were estimated from transfer function curve fits to gain and phase data obtained from pseudorandom rotation test results. Solid curves are lowess fits.

Fig. 11. OKR gain constant (A), time constant (B), and time delay (C) parameters as a function of subject age. Parameter values were estimated from transfer function curve fits to OKR gain and phase data obtained from pseudorandom rotation test results. Solid curves are lowess fits.

Fig. 12. Caloric test peak slow phase eye velocity (A) and unilateral weakness (B) as a function of subject age. Solid curves are lowess fits.

Fig. 13. Comparison of age-related changes in VOR gain and peripheral vestibular anatomical data. The 0.8 Hz VOR gain fit is the same as in Figure 9. All curve fits to anatomic data are lowess fits to published data. All fits are plotted on a linear scale normalized to 1.0 at age 30 years. The normalization factors are 0.87 for 0.8 Hz VOR gain, 6940 crista hair cells, 17450 vestibular nerve fibers, and 18135 Scarpa's Ganglion cells.

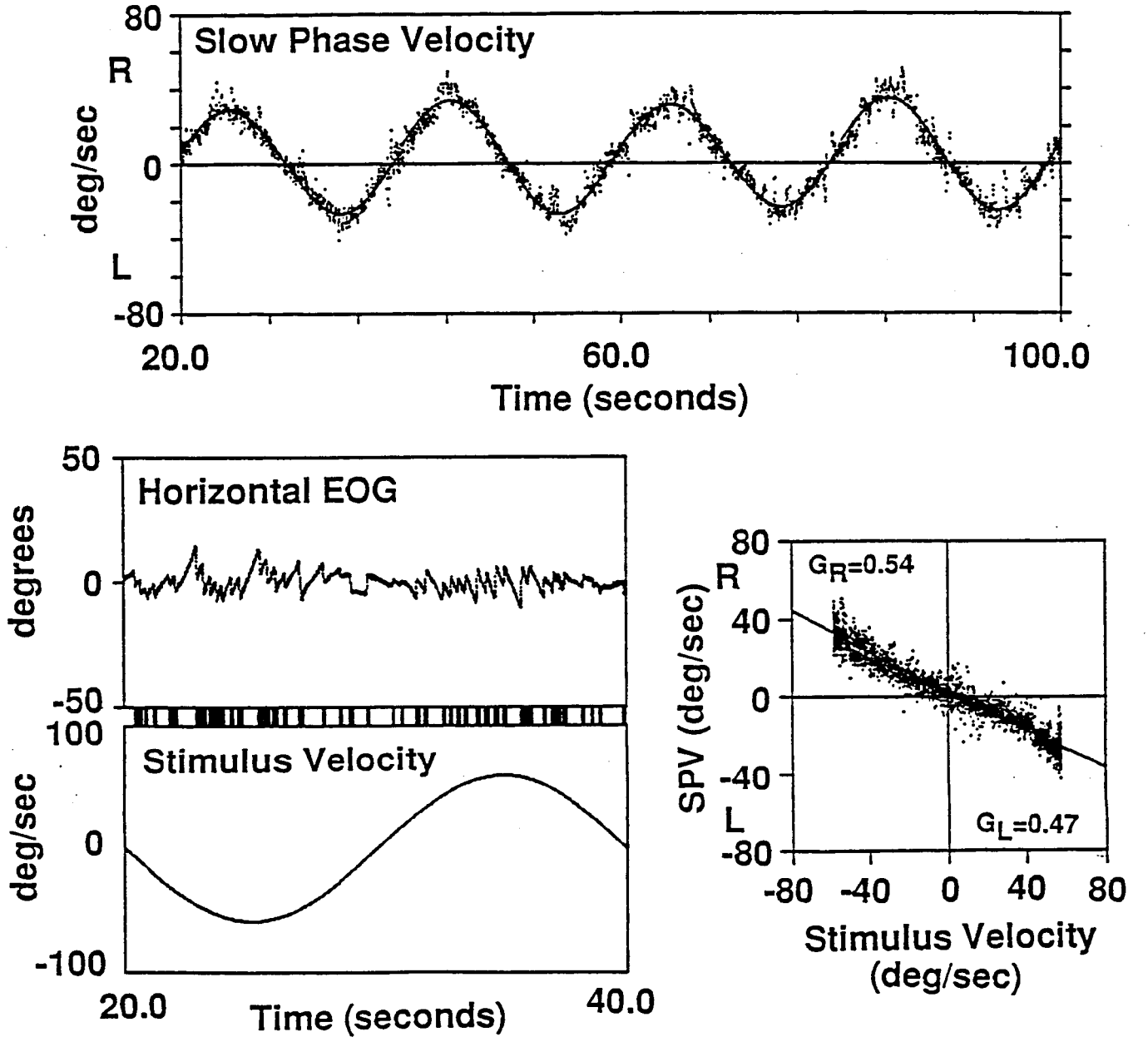

Slow Phase Velocity
80
R
0
L
-80
deg/sec
20.0
60.0
100.0
Time (seconds)
Horizontal EOG
50
0
-50
degrees
Stimulus Velocity
100
0
-100
deg/sec
20.0
Time (seconds)
40.0
SPV (deg/sec)
G_R=0.54
G_L=0.47
-80 -40 0 40 80
Stimulus Velocity
(deg/sec)

Figure 1

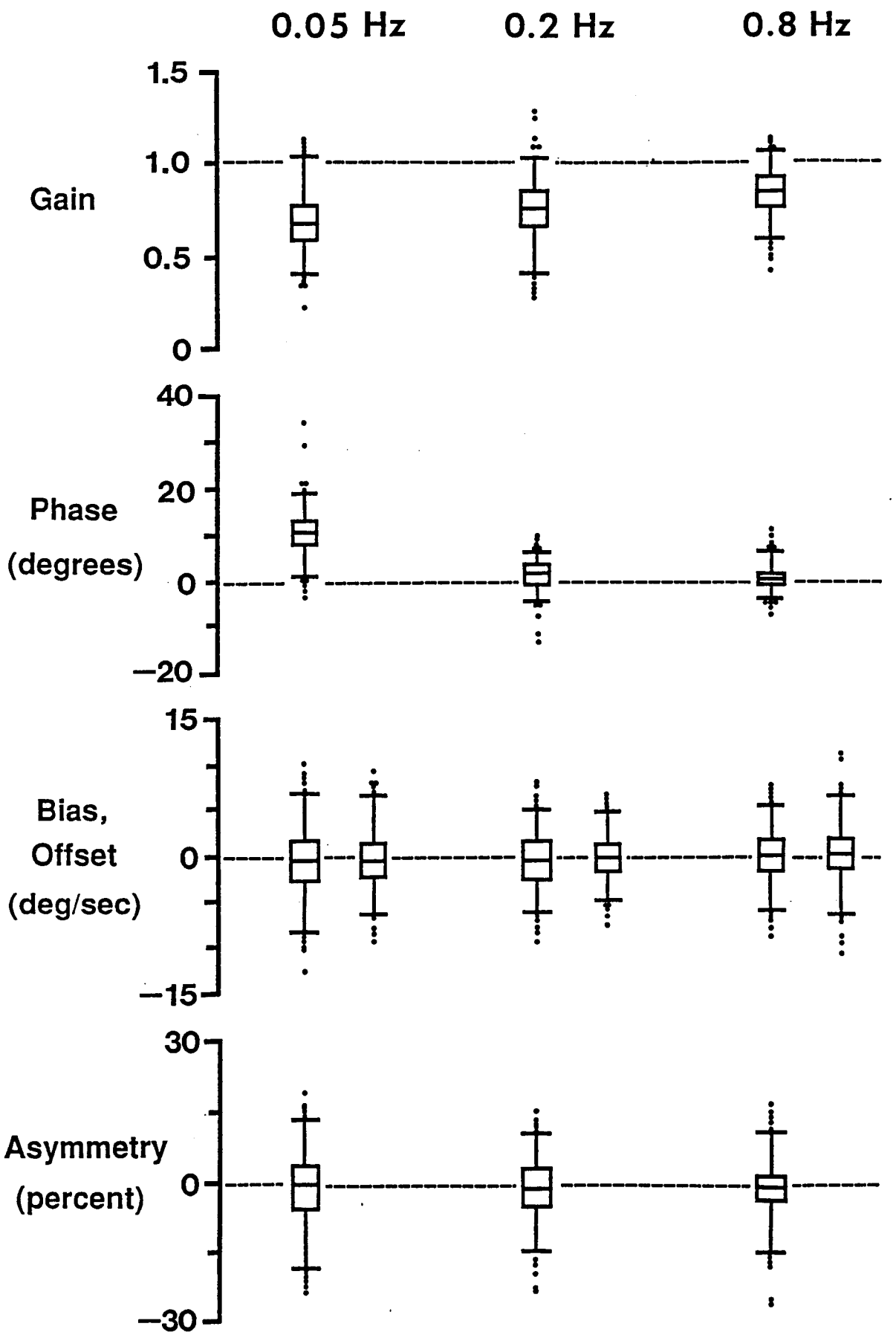

0.05 Hz
0.2 Hz
0.8 Hz
Gain
1.5
1.0
0.5
0
Phase
(degrees)
40
20
0
−20
Bias,
Offset
(deg/sec)
15
0
−15
Asymmetry
(percent)
30
0
−30

Figure 2

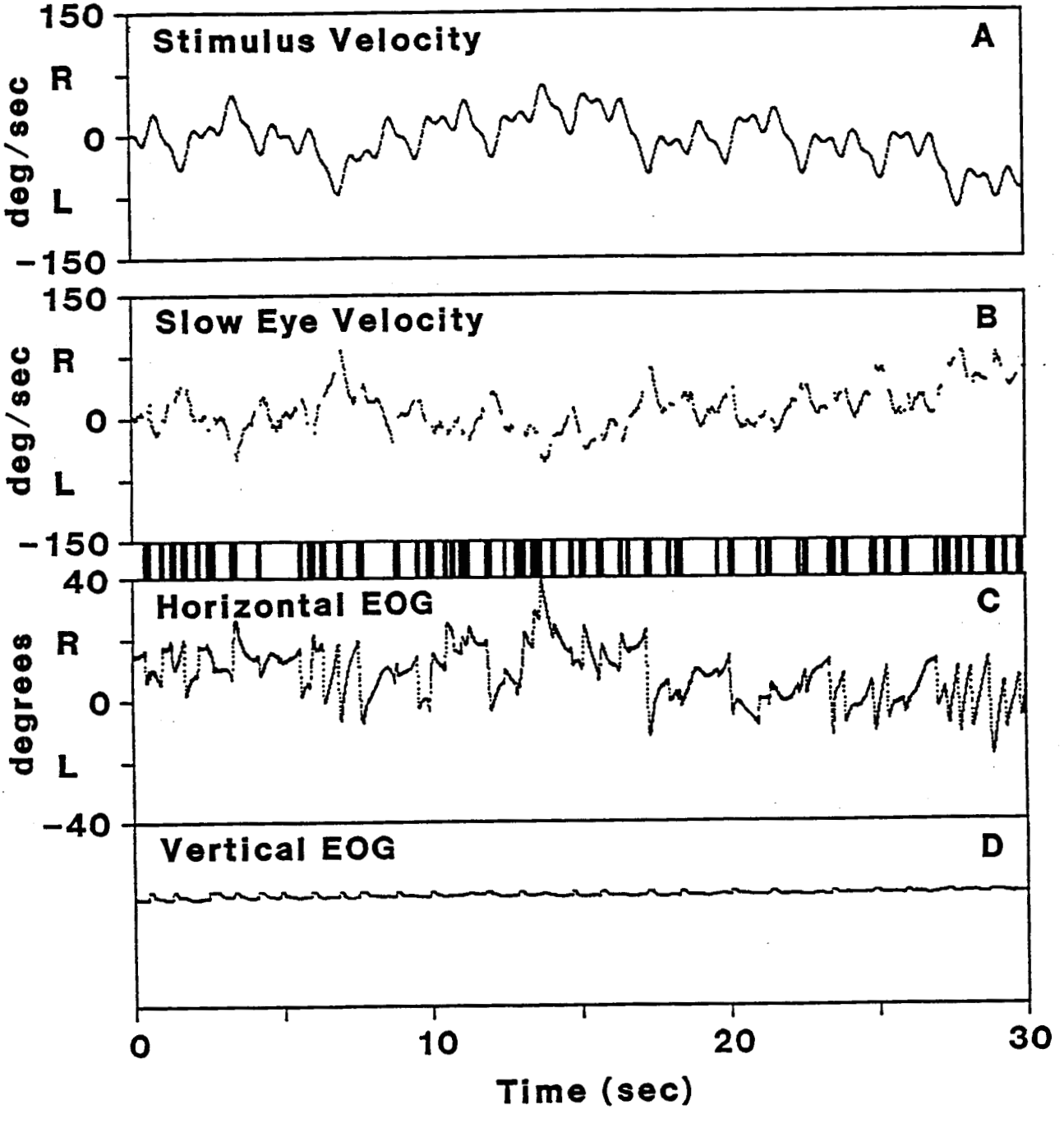

150
R
0
L
-150
deg/sec
Stimulus Velocity
A
150
R
0
L
-150
deg/sec
Slow Eye Velocity
B
40
R
0
L
-40
degrees
Horizontal EOG
C
Vertical EOG
D
0
10
20
30
Time (sec)

Figure 3

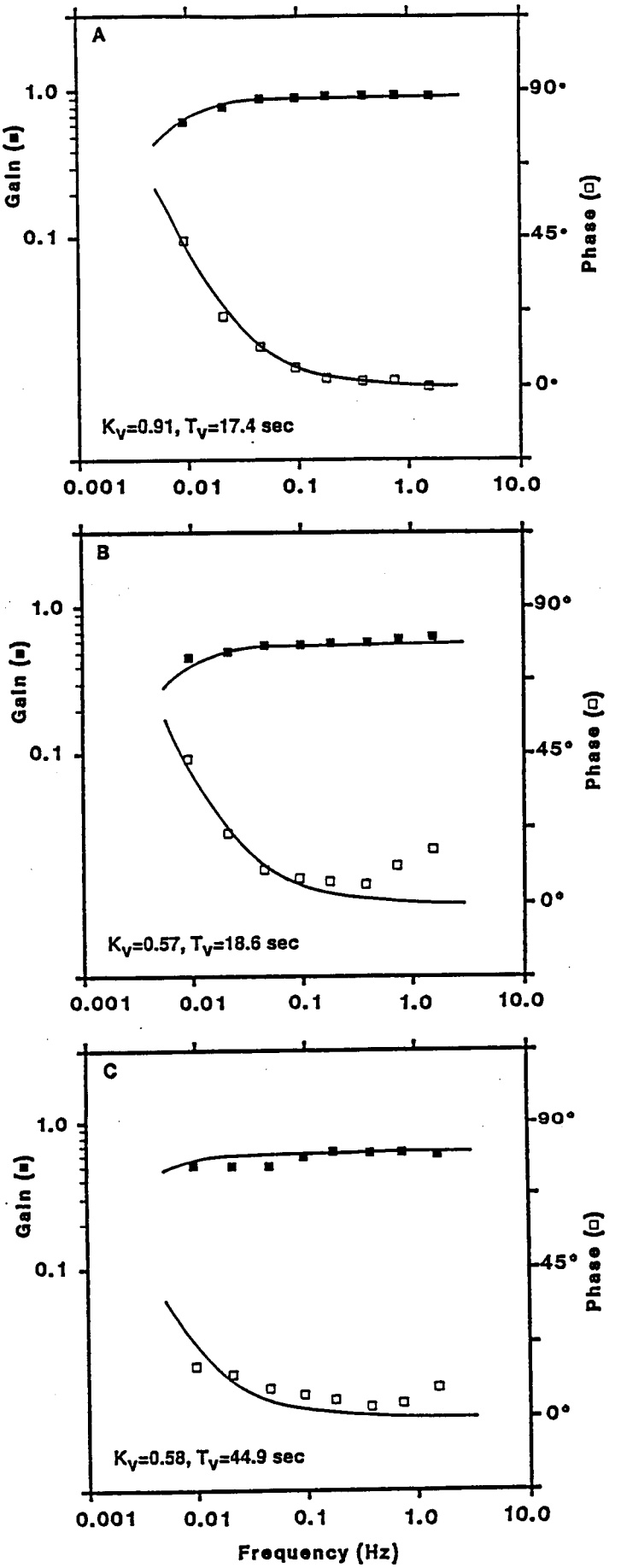

A
1.0
0.1
Gain (■)
90°
45°
0°
Phase (□)
K_V=0.91, T_V=17.4 sec
0.001
0.01
0.1
1.0
10.0
B
1.0
0.1
Gain (■)
90°
45°
0°
Phase (□)
K_V=0.57, T_V=18.6 sec
0.001
0.01
0.1
1.0
10.0
C
1.0
0.1
Gain (■)
90°
45°
0°
Phase (□)
K_V=0.58, T_V=44.9 sec
0.001
0.01
0.1
1.0
10.0
Frequency (Hz)

Figure 4

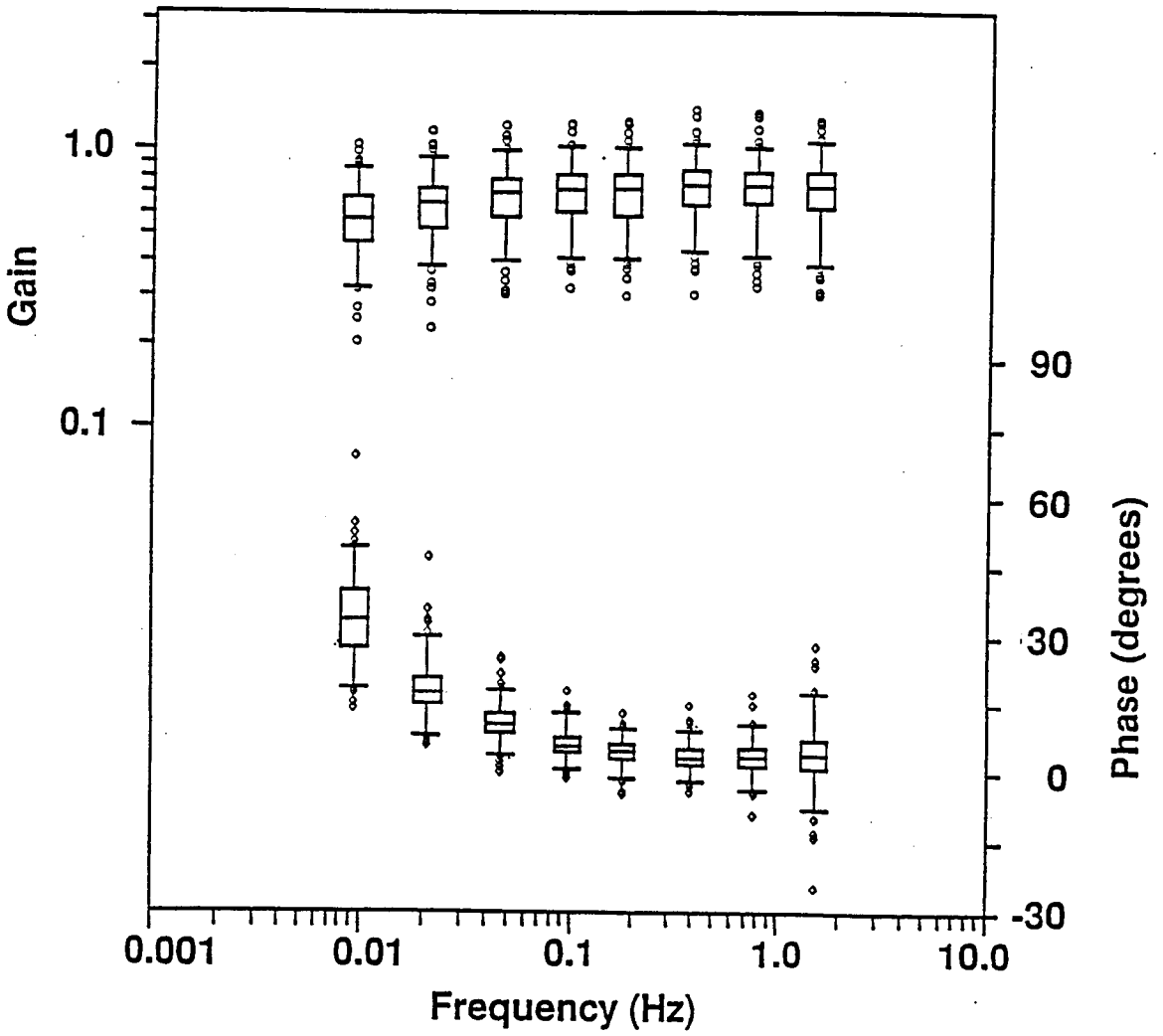

Gain
1.0
0.1
90
60
30
0
-30
Phase (degrees)
0.001
0.01
0.1
1.0
10.0
Frequency (Hz)

Figure 5

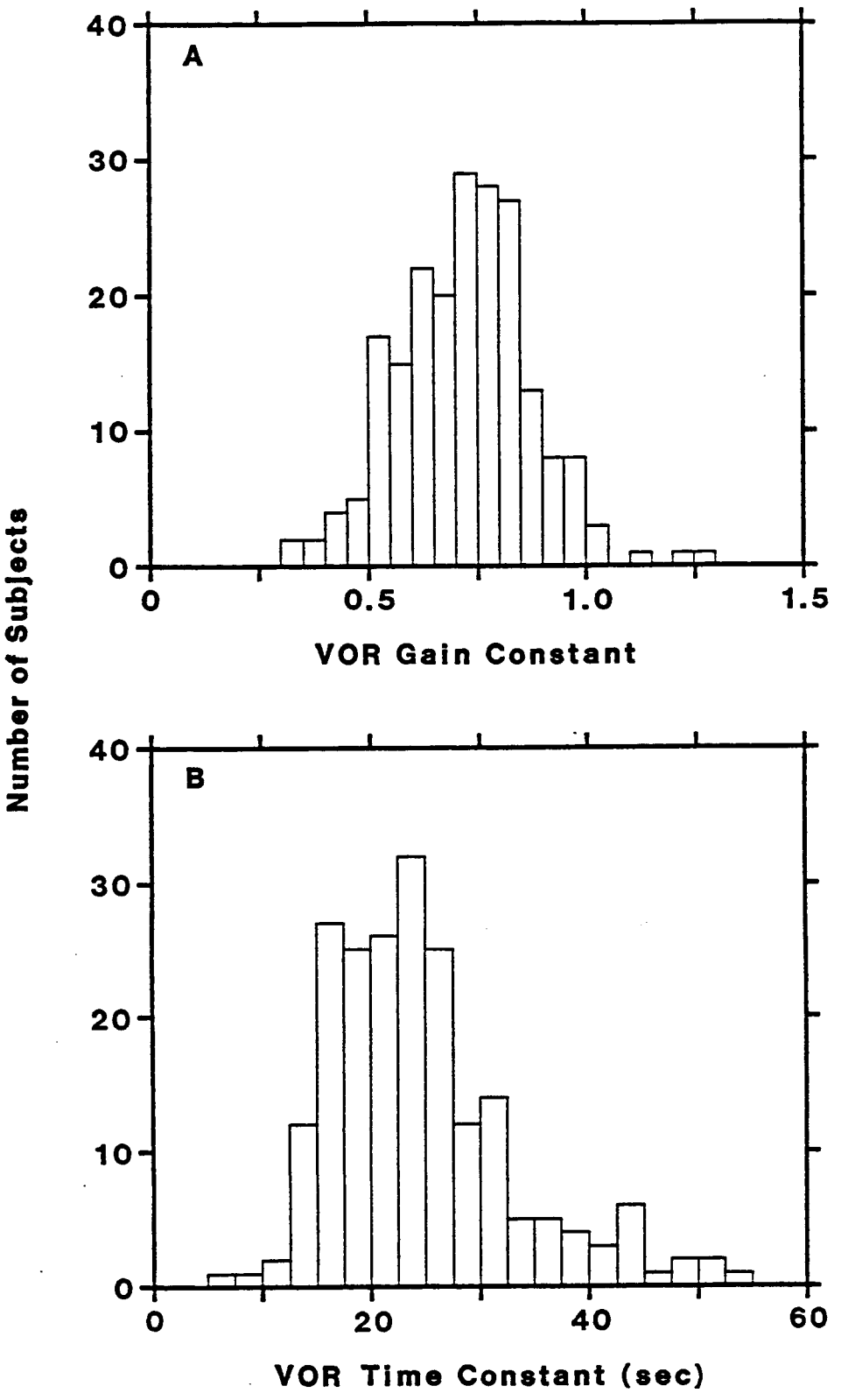

A
B
40
30
20
10
0
0
0.5
1.0
1.5
60
Number of Subjects
VOR Gain Constant
VOR Time Constant (sec)

Figure 6

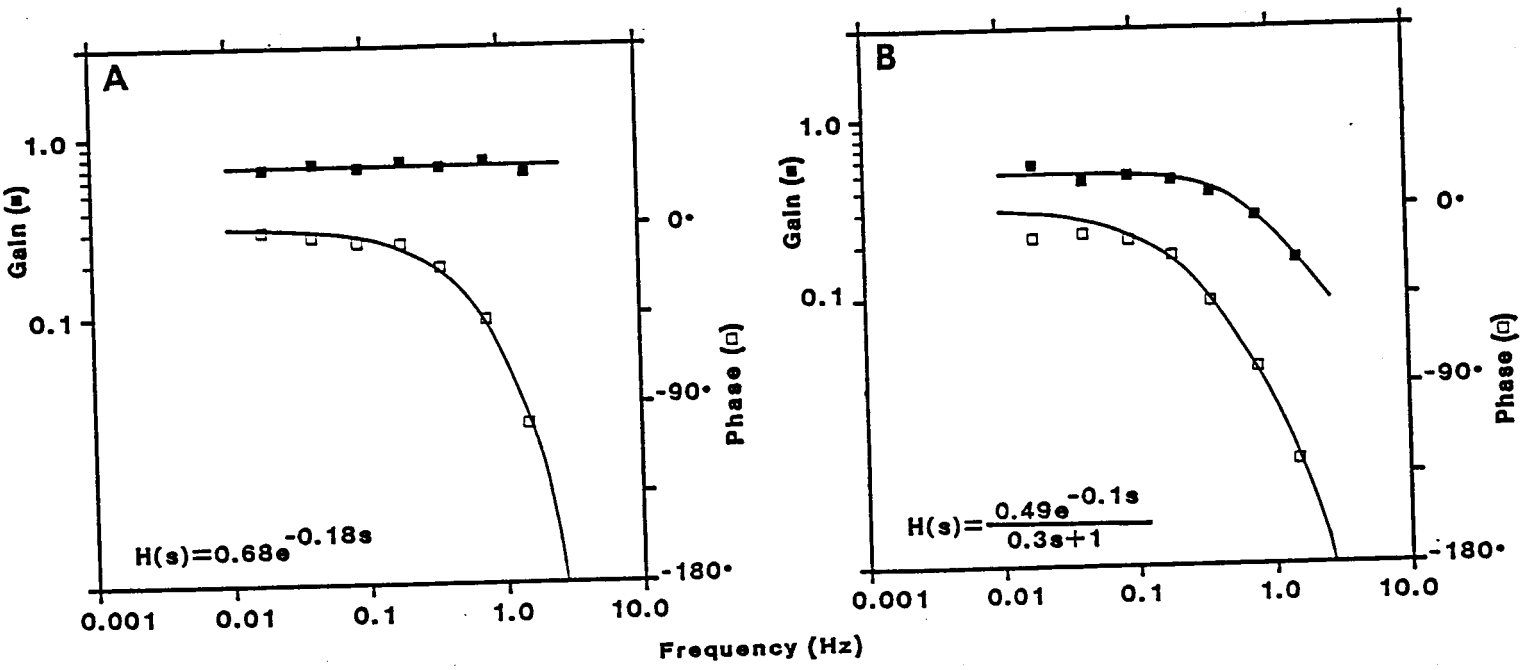

A
B
Gain (■)
Phase (□)
1.0
0.1
0°
-90°
-180°
0.001
0.01
0.1
1.0
10.0
Frequency (Hz)
H(s)=0.68e^-0.18s
H(s)=0.49e^-0.1s / 0.3s+1

Figure 7

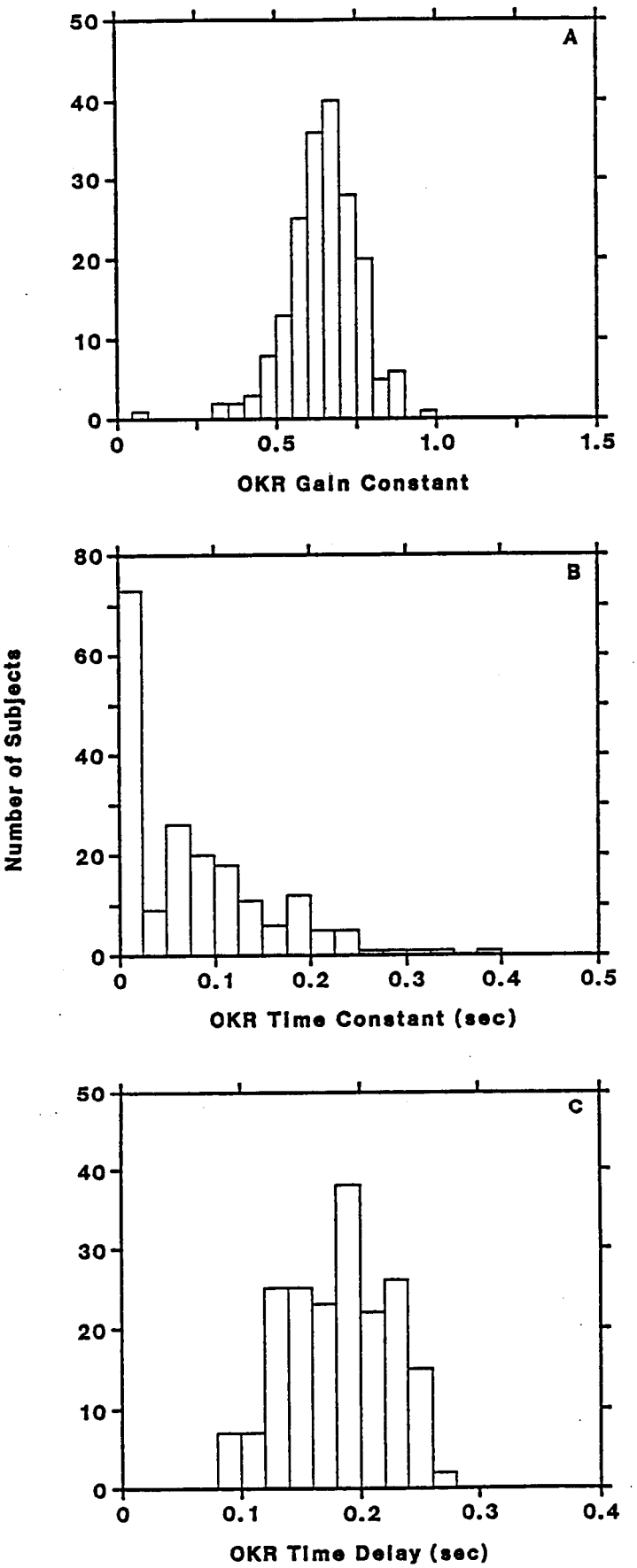

A
50
40
30
20
10
0
0
0.5
1.0
1.5
OKR Gain Constant
B
80
60
40
20
0
0
0.1
0.2
0.3
0.4
0.5
OKR Time Constant (sec)
Number of Subjects
C
50
40
30
20
10
0
0
0.1
0.2
0.3
0.4
OKR Time Delay (sec)

Figure 8

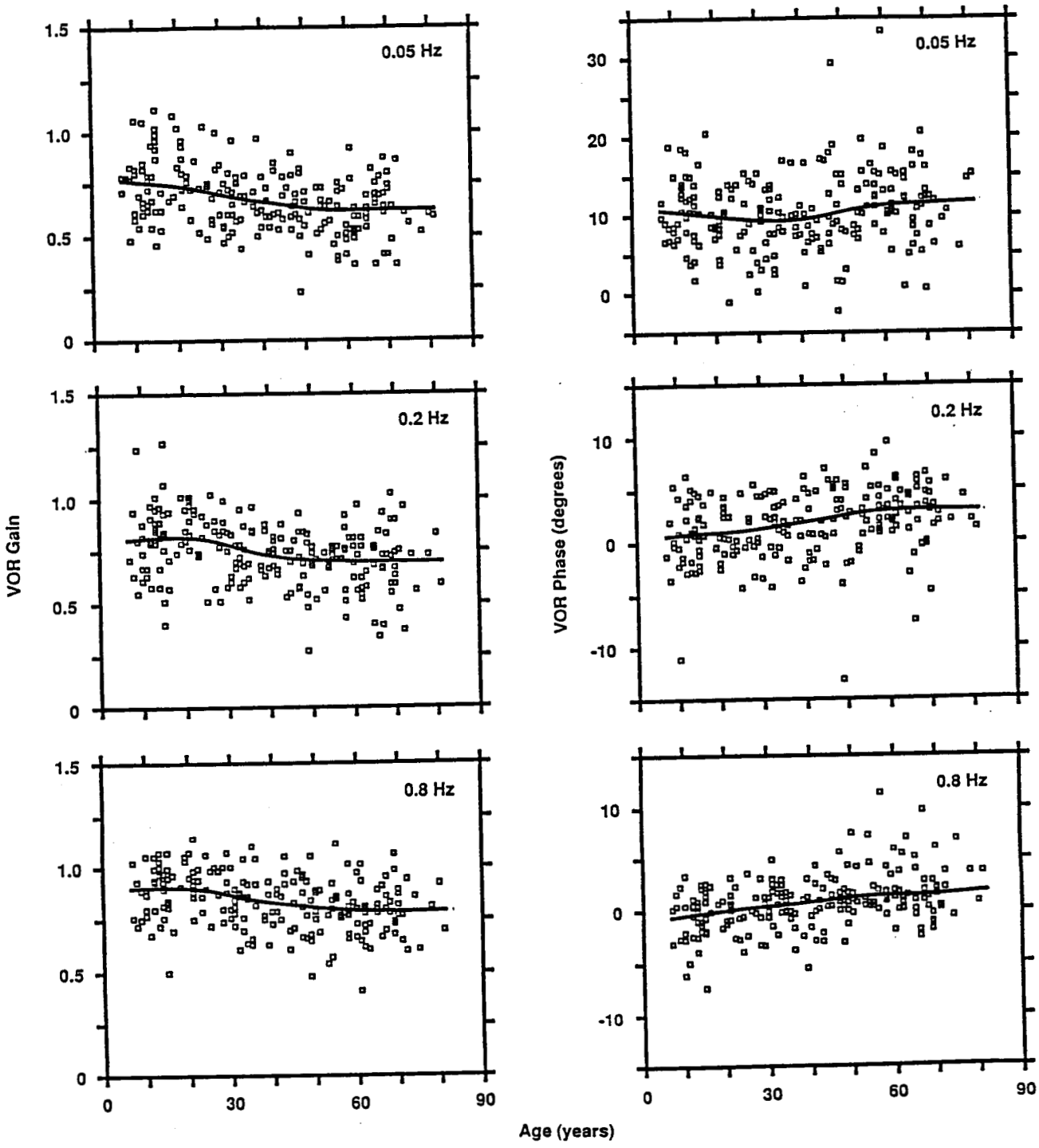

0.05 Hz
0.2 Hz
0.8 Hz
VOR Gain
VOR Phase (degrees)
Age (years)
1.5
1.0
0.5
0
30
20
10
-10
0
30
60
90

Figure 9

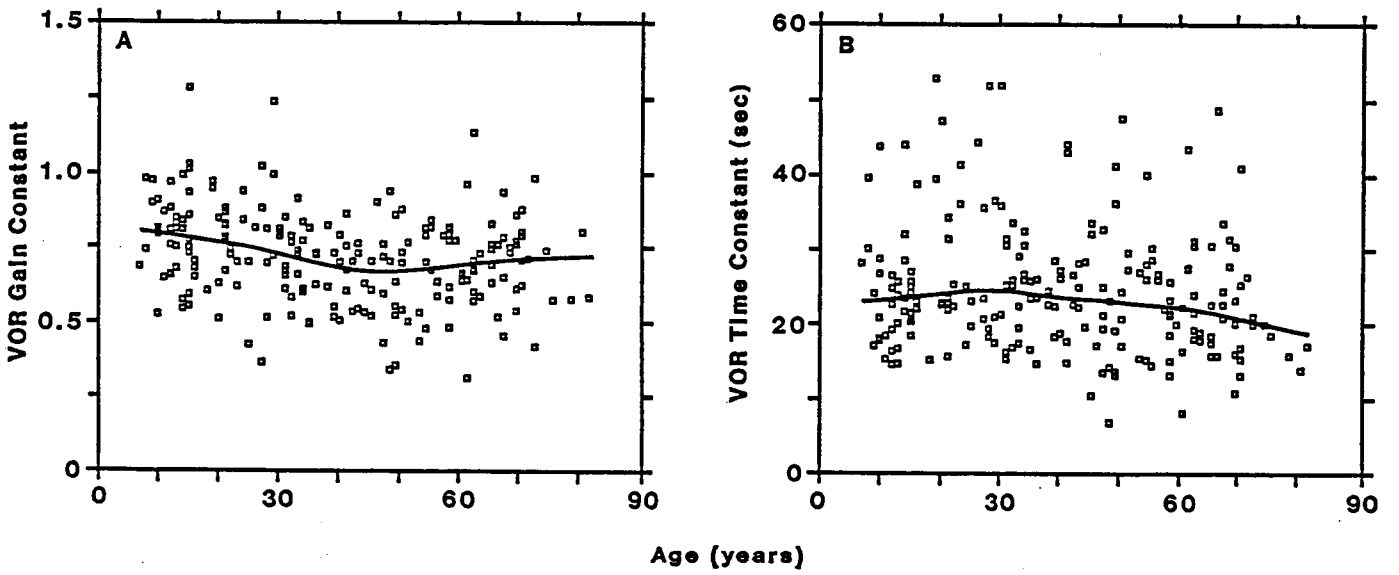

A
B
VOR Gain Constant
VOR Time Constant (sec)
1.5
1.0
0.5
0
60
40
20
0
30
60
90
Age (years)

Figure 10

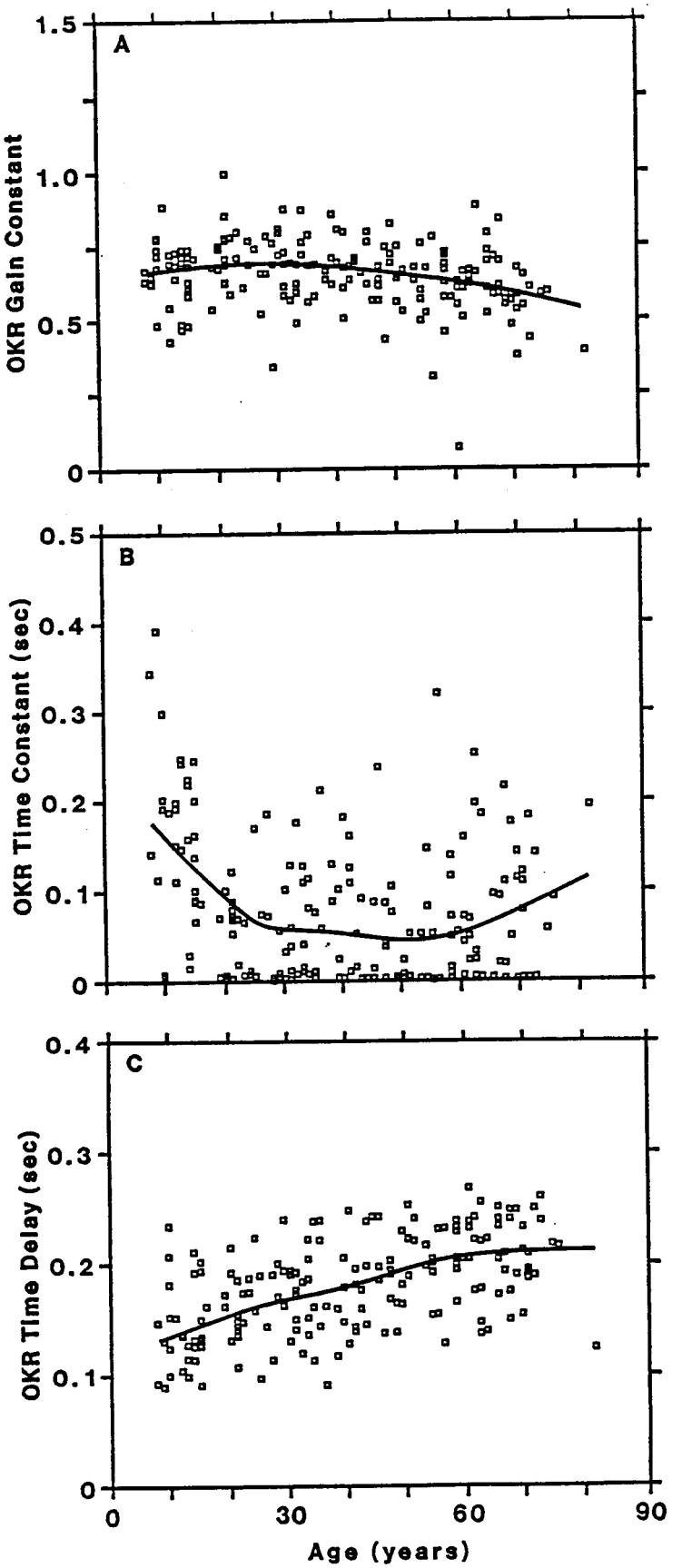

A
OKR Gain Constant
1.5
1.0
0.5
0
B
OKR Time Constant (sec)
0.5
0.4
0.3
0.2
0.1
0
C
OKR Time Delay (sec)
0.4
0.3
0.2
0.1
0
0
30
60
90
Age (years)

Figure 11

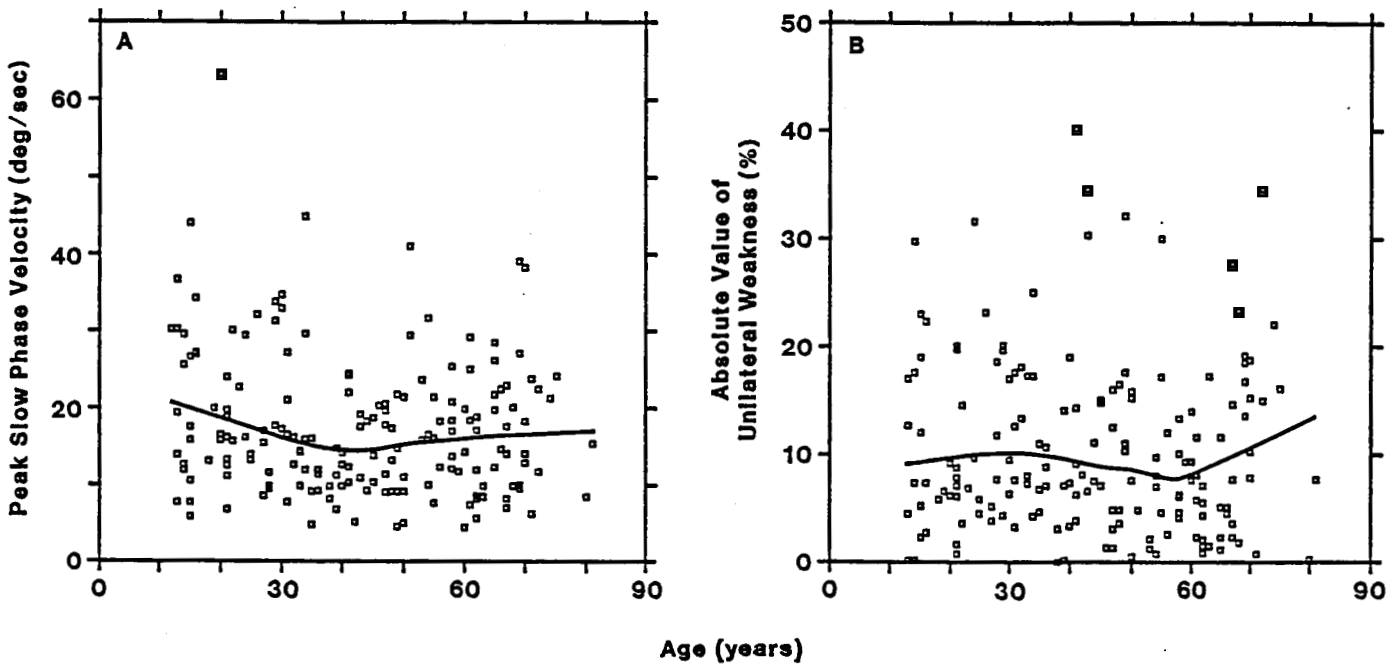

A
B
Peak Slow Phase Velocity (deg/sec)
Absolute Value of Unilateral Weakness (%)
0
20
40
60
10
30
50
0
30
60
90
Age (years)

Figure 12

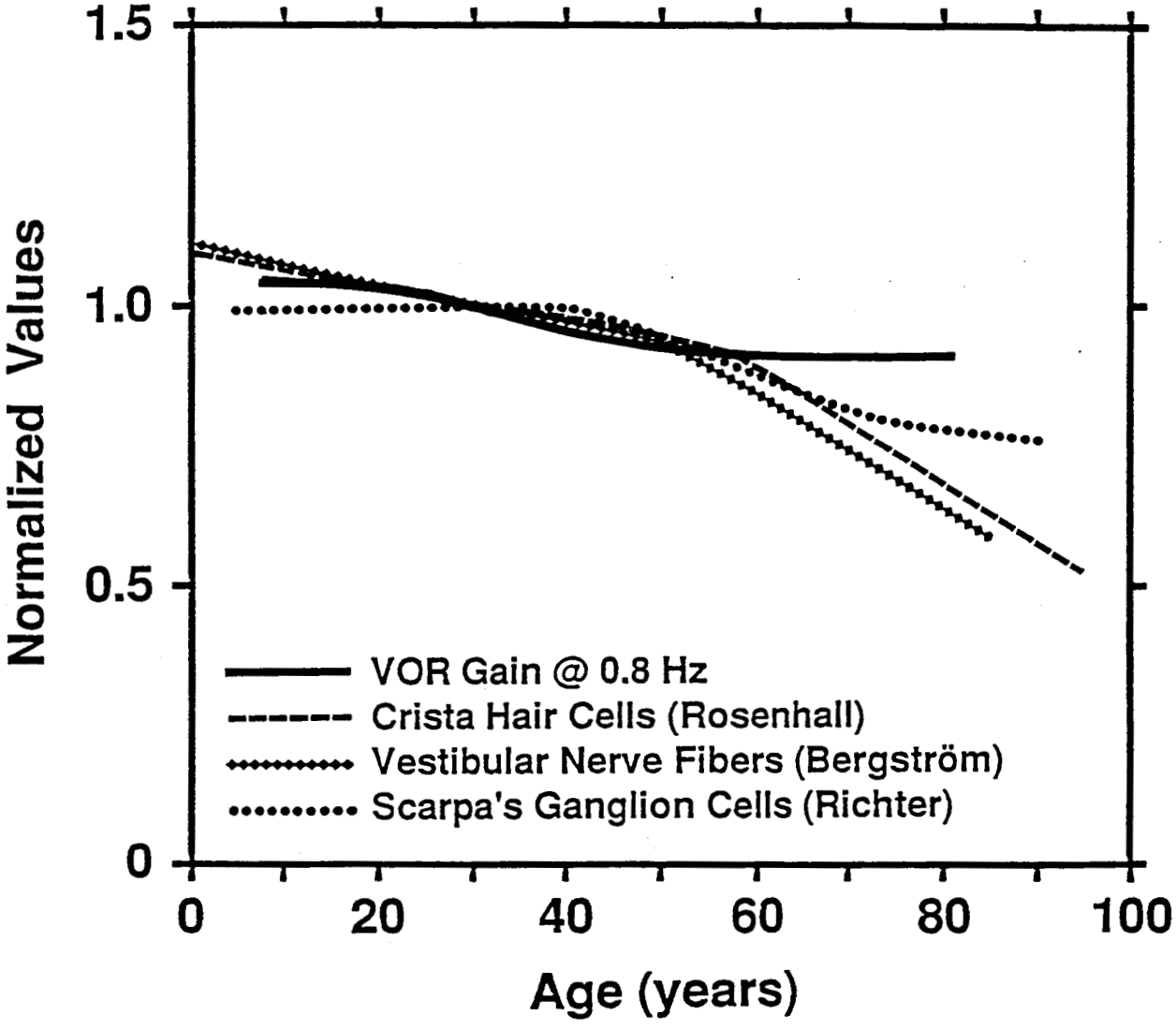

1.5
1.0
0.5
0
Normalized Values
0
20
40
60
80
100
Age (years)
VOR Gain @ 0.8 Hz
Crista Hair Cells (Rosenhall)
Vestibular Nerve Fibers (Bergström)
Scarpa's Ganglion Cells (Richter)

Figure 13

AGE-RELATED CHANGES IN HUMAN POSTURE CONTROL

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Summary.

Posture control responses were measured in 214 human subjects ranging in age from 7 to 81 years. Motor control tests measured various leg muscle EMG latencies in response to sudden forward and backward horizontal translations of the support surface upon which the subjects stood. EMG latencies increased with increasing age. The rate at which latency increased with age was larger for subjects over 55 years. Sensory interaction tests measured the magnitude of sway during six 20 second trials in which visual and proprioceptive orientation cues were altered (by rotating the visual surround and support surface in proportion to the subject's sway) or vision eliminated (eyes closed) in various combinations. No age-related decline in postural stability was found for subjects standing on a fixed support surface with eyes open or closed. However, age-related declines in stability were found for conditions involving altered visual or proprioceptive sensory cues. Subjects older than about 55 years showed the largest performance decline. Subjects younger than about 15 years were also sensitive to alteration of sensory cues. On average, the older subjects were more affected by altered visual cues while younger subjects had difficulty with altered proprioceptive cues. About 30% of subjects older than 50 years fell on two or three of the four sensory tests with altered visual and/or proprioceptive cues. There were two main patterns of falls. One group fell only when visual cues were altered but performed normally when vision was absent suggesting that this group relied on visual orientation cues whenever available, even though the visual cues conflicted with proprioceptive and/or vestibular cues. The other group fell when proprioceptive cues were altered and visual cues were either altered or absent suggesting that they were not able to use vestibular cues for posture control, or that their peripheral vestibular function was reduced.

Key Words: Posture - Motor Control - Aging - Humans

Introduction

The automatic control of upright stance is an active sensorimotor feedback process which must keep the body's center of gravity over the base of support (the feet). This process requires that deviations of body position from upright must be sensed and then processed to initiate motor commands which oppose the initial deviation and return the body to an upright position. The vestibular, proprioceptive, and visual systems are the main sources of sensory information on body motion. However situations commonly arise in which information from the various sensory systems is absent, altered, or distorted even in individuals with normal sensory function. For example, proprioceptive cues can be misleading when standing on a compliant surface, and visual cues are eliminated in darkened rooms or when the eyes are closed. In addition, vestibular information about body sway must be processed in a way which accounts for the position of the head on the body and in space since the pattern of activity generated in the various semicircular canals and otolith organs depends on their orientation. Motion information from these various sensory systems must be combined in order to generate motor control signals for the appropriate muscle groups with the correct direction, amplitude, and timing. Finally the forces generated by the muscle fiber contractile elements must act through the dynamic mechanical elements in the muscles, tendons, and joints to produce motion which corrects for the initial deviation in body position. Posture control is therefore a complex task requiring a high degree of sensorimotor integration.

Two general methods have been used to study human postural control mechanisms. They are distinguished mainly by the time course of the posture control responses elicited by the various experimental conditions. The first method, which we will call motor control tests, concerns postural adjustments which occur in the first second following a perturbation of the body. The second method, which we will call sensory interaction tests, involves posture responses which occur over tens of seconds to minutes in response to various sensory conditions. Both methods typically correlate recordings of forces or torques exerted on the support surface, body sway angles, center of pressure, and leg and trunk muscle EMG's with various experimental conditions.

Motor control tests typically measure postural reactions to short duration translations or rotations of the support surface (Nashner 1977; Allum 1983; Diener et al. 1983). Various factors have been shown to influence these short term reactions. These factors include support surface condition (Horak and Nashner 1986), initial body position (Diener et al. 1983; Moore et al. 1986), stimulus velocity and displacement amplitudes (Diener et al. 1988), galvanic stimulation (Nashner and Wolfson 1974), and availability of visual (Nashner and Berthoz 1978) and proprioceptive cues (Diener et al. 1984).

The simplest sensory interaction tests contrast spontaneous postural movements when the subject's eyes are open and closed (Murray et al. 1975; Black et al. 1982). In clinical settings these are referred to as standard Romberg tests. Other experiments have investigated the effects of external disturbances on posture. These disturbances have included altered visual sensory cues using moving visual fields (Lestienne et al. 1977; Clement et al. 1985), altered proprioceptive cues using calf muscle vibration (Pyykko et al. 1983; Johansson et al. 1988), compliant surfaces (Amblard et al. 1985), and support surface translations (Maki et al. 1987; Ishida and Imai 1980), or

altered vestibular cues by changing head position (Brandt et al. 1981) or galvanic stimuli (Watanabe et al. 1985). Another variation alters proprioceptive and visual motion cues by rotating the subject's visual field and/or support surface in equal proportion to the subject's own sway (Nashner 1971; Vidal et al. 1978; Nashner et al. 1982).

The complexity of maintaining upright stance suggests that there would be a great deal of functional variability within a normal population as a result of variations in sensory system, central nervous system, and biomechanical function in individuals. In addition, the increased incidence of falls in the older population (Sheldon 1960; Overstall 1978; Prudham and Evans 1981; Sixt and Landahl 1987) suggests that one or more of the components required for accurate posture control degenerates with age. Studies which have looked for differences in posture control between young and old adults have generally found them (Fregly et al. 1973; Murray et al. 1975; Woollacott et al. 1986). These changes include increased sway or falls during various sensory interaction tests, and slightly increased response latencies in motor control tests. In addition children show developmental changes in posture control which converge to adult patterns at about age 10 years (Forssberg and Nashner 1982; Riach and Hayes 1985). However the limited scope of these studies with their small sample sizes and restricted test paradigms have not clarified either the time course or the mechanisms involved in the changes in posture control with increasing age. We tested a putatively normal population with a wide age distribution using both motor control and sensory interaction tests. In addition, vestibulo-ocular and oculomotor reflexes were independently tested in the same individuals (Peterka et al. 1989) for comparison to posture control responses.

Methods (Note: Methods section should be in small print)

Posture control function was tested in 214 human subjects (90 male and 124 female) aged 7 to 81 years. Ages were approximately uniformly distributed over the entire range. Tests included 1) motor control tests with short duration forward and backward horizontal translations of the surface upon which the subjects stood, and 2) sensory interaction tests which presented various combinations of normal and altered proprioceptive and visual cues which might affect posture control. The horizontal vestibulo-ocular and optokinetic reflexes were measured in these same subjects on the same day, and are reported in a companion paper (Peterka et al. 1989).

Subjects were required to meet the following criteria:

1. normal age-corrected auditory pure tone responses
2. middle ear reflexes present bilaterally
3. normal middle ear impedance
4. no history of head blows of sufficient magnitude to cause loss of consciousness
5. no abnormal neurological findings
6. normal corrected vision
7. no history of use of ototoxic drugs
8. no history of dizziness or disequilibrium
9. moderate or absent use of alcohol
10. no use of psychotropic drugs

Subjects were not excluded from the population based on any vestibular, oculomotor, or posture test results.

Subjects stood on a movable support surface surrounded in front and on two sides by a visual surround which could also move. The visual surround was a box with randomly placed 2 cm black dots on a flat white surface. The average spacing between the dots was about 20 cm, and the distance from the subject to the box was about 50 cm. Support surface motion was controlled by a hydraulic position servo system which could produce toe up and toe down rotations about an axis collinear with the subject's ankle joints and forward and backward translations. Visual surround motion was controlled by a separate hydraulic servo system which rotated the box about the ankle joint axis. Force transducers in the support surface recorded forces and torques applied by each of the subjects' legs. The anterior-posterior (AP) sway angle (θ_{ap}) of each subject was recorded using a rod attached to a potentiometer. The potentiometer was mounted on a post next to the subject. The end of the rod rested in a V-shaped holder centered on the subject's back at hip level. A voltage proportional to the angular displacement of the potentiometer was recorded and later transformed using appropriate trigonometric conversions to θ_{ap} (Figure 1). A second potentiometer mounted at shoulder level recorded AP displacements at the shoulder in the last 65 subjects tested. A measure of hip angle (θ_h, Figure 1) was calculated from AP angles measured at the hip and shoulder. An approximate center-of-gravity AP sway angle (θ_{cg}) was calculated using the following formula:

$$\theta cg = \tan^{-1}\left[\frac{0.860\sin\theta_{ap} + 0.242\sin(\theta_{ap}+\theta_h)}{0.860\cos\theta_{ap} + 0.242\cos(\theta_{ap}+\theta_h)}\right]$$

This formula was derived assuming the subjects had average body mass distribution and average proportional lengths of various body segments (Diffrient et al. 1974). To the extent that the various subjects deviated from average body configurations, the measurement of θ_{cg} would be in error. This error was probably not more than 10% in this population. Measurements of forces, torques, and sway angles were sampled at 50 Hz and saved for later analysis.

Upright stance without moving the feet can be achieved using two different body motion strategies (Horak and Nashner 1986). A hip strategy consists of θ_h and θ_{ap} motions which are out of phase. Subjects can be forced to use a hip strategy by asking them to stand on a narrow beam where ankle muscles cannot exert torque about the ankle joint. A pure ankle strategy occurs when all motion is about the ankle joint (AP sway angles measured at the hip and shoulder are equal and θ_h is zero). A less pure ankle strategy occurs when there is some motion about the hip joint, but θ_{ap} and θ_h are in phase with each other. In order to quantify the type of body motion, a strategy measure was calculated according to the following formula:

$$\text{strategy score} = \overline{[(\theta_{ap}-\bar{\theta}_{ap})(\theta_h-\bar{\theta}_h)]}$$

where the bars over the various terms indicate the average values. In words, the strategy score is the average cross product of zero-meaned θ_{ap} and θ_h calculated over the duration of the trial. The strategy score is negative if θ_h and θ_{ap} are out of phase, positive if they are in phase and the body moves

like a whip, and zero when the body moves like an inverted pendulum with no bending at the waist. Since this measure is an average over the entire trial, changes in strategy during the trial would not be correctly characterized by this single measure. In practice this was not a problem since this putatively normal population did not show marked strategy changes within trials.

Motor Control Tests

Motor tests consisted of five each of forward translations, toe down rotations, backward translations, and toe up rotations of the support surface on which the subject stood with eyes open viewing the stationary visual surround. Ramp translations were 3 cm in 0.25 s and rotations were 5^{0} in 0.25 s. The support surface returned slowly to the center position following each motion and there was a variable delay averaging 4 s between stimuli. In addition to the forces, torques, and sways described above, four EMG's were recorded from the left leg using surface electrodes over the gastrocnemius (G), tibialis anterior (T), hamstring (H), and quadriceps (Q) muscles. EMG's were rectified, low pass filtered at 20 Hz, and sampled at 500 Hz. The latency to the onset of the reflex EMG bursts were estimated from average EMG traces and were referenced to the beginning of support surface motions. Onset times estimated in this way were probably biased toward the shortest onset time of the five individual trials. Latencies were recorded from averaged traces only if the EMG onset times could unambiguously be separated from background activity. Only responses to translations are reported in this paper.

Sensory Interaction Tests

The sensory tests provided a functional evaluation of the ability of subjects to effectively use vestibular, proprioceptive, and visual information in the control of their upright posture (Nashner et al. 1982). The subjects' task was to maintain an upright stance for 21 seconds with as little postural sway as possible while they were presented with six different sensory conditions.

Conditions 1 and 2 required the subjects to stand on a stable surface for 21 seconds facing an earth-fixed visual field with eyes open and then with eyes closed. The remaining four conditions placed the subject in more demanding sensory environments. These environments were created by rotating the visual field and/or the support surface in equal proportion to θ_{ap}. For example, as the subject swayed forward, the visual field rotated forward about an axis through the ankle joint. Under this condition the subject saw little or no change in orientation of the visual field with respect to himself. This is referred to as sway-referenced vision as opposed to the earth-referenced vision in condition 1. The same technique was applied to the support surface by rotating it about the ankle joint in proportion θ_{ap}. This sway-referenced support condition resulted in little or no change in ankle joint angle as the subject swayed forward and back and therefore altered the ankle joint proprioceptive cues contributing to posture control. The entire sensory test sequence included all six combinations of eyes closed, sway-referenced, and earth-referenced vision and support surface conditions given in Table 1.

Sway angles were quantified by two measures: average rectified sway and peak-to-peak sway. Both measures were calculated over the final 20 seconds

of the 21 second trials. The visual field and support surface were always earth-referenced during the first second of each trial. The sway data was normalized by subtracting the average sway values recorded in the first second from the entire sway record. Average rectified sway often did not reflect how close a given individual was to a fall since, for example, a subject who leaned forward by a few degrees and stayed in that position throughout the remainder of the trial could score the same as a subject who oscillated back and forth during the trial with the peak of the oscillations being close to the threshold of a fall. Therefore a measure of peak-to-peak sway was also used to characterize performance.

In order to visualize trends in various scatterplots, a robust locally weighted regression analysis (lowess fit) was used to smooth the scatterplots (Cleveland 1979, 1985). A lowess smoothing parameter of 0.5 and iteration parameter of 2 were used on all data sets.

Results

Motor Control Tests

The pattern of EMG responses to rapid forward and backward translations of the support surface have been described previously (Nashner 1977). Distal leg muscle activation generally precedes proximal leg muscle activation. For example, backward support surface translations result in forward body sway. EMG responses from the gastrocnemius muscle group on the back of the leg begin about 90 ms after the start of the translation. The hamstring EMG begins about 20-30 ms after the gastrocnemius. A similar pattern of muscle activity occurs for forward translations with the tibialis EMG beginning about 90 ms after the start of the translation. The start of the quadriceps EMG typically follows the onset of tibialis EMG by about 20 ms.

Figure 2 shows a typical EMG response pattern for one typical subject. Because of the response variability it was often difficult to judge the EMG onset from a single trial. Therefore EMG latencies were measured from EMG responses averaged over 5 trials. The population distributions of EMG onset times were fairly symmetric with means of 90 ms (12.7 ms s.d.) and 119 ms (21.6 ms s.d.) for G and H during backward translations, and 89 ms (12.8 ms s.d.) and 105 ms (21.6 ms s.d.) for T and Q during forward translations. These values are similar to those reported by Horak and Nashner (1986) who used similar stimulus conditions.

The plots of G versus H and T versus Q onset times in Figure 3 indicates that there is a great deal of individual variability in the delay between proximal and distal muscles onsets. The lowess fit to the G and H delays is roughly parallel to but offset from the line of equal H and G onset times (dashed line). This indicates that on average the delay between G and H is simply a constant of about 30 ms which is independent of the G onset value. In contrast the lowess fit to the T and Q delays is sloped in toward the line of equal T and Q onset times. This indicates that on average subjects with larger T onset times will have a shorter delay between the start of the T and the start of the Q muscle bursts than subjects with a shorter T onset time. Linear correlation coefficients were $r=0.555$ and 0.282 for H versus G and Q versus T, respectively.

Age-Related Changes in Motor Test Results

With the exception of the Q muscle, EMG onset times generally increased with increasing subject age (Figure 4). Linear fits to the data showed that the rate of change of EMG onset times with age were 0.28, 0.50, 0.16, and -0.02 ms/year with linear correlation coefficients of r=0.444, 0.433, 0.259, and -0.022 for G, H, T, and Q respectively. However the lowess fits to the G, T, and H EMG onset times suggested that there may be an inflection point at about age 55 with a larger rate of change for subjects older than 55 years. To compare the rates for younger and older subjects, two part linear fits were made to G, H, and T onset times for subjects younger and older than 55 years with the constraint that the two linear fits intersect at age 55 years. The slopes were 0.26, 0.45, and 0.09 ms/year for younger subjects, and 0.37, 0.67, and 0.48 ms/year for the older subjects for G, H, and T respectively. Therefore these two part linear fits reveal the same trend as the lowess fits. The slowing of motor responses with increasing age was most evident in the T responses where the rate of change of T onset times with age was more than 5 times larger in the older group.

The difference between the EMG onset times for the H and G muscles (H-G) during backward translations, and between Q and T muscles (Q-T) during forward translations is plotted as a function of subject age in Figure 5. There was a slight increase in the H-G delay with increasing age (0.17 ms/year with r=0.185). For the Q-T delay, subjects younger than about 17 years tended to have larger Q-T delays (mean 26.8 ms +/- 20.2 s.d.) than subjects older than 17 years (mean 13 ms +/- 23.9 s.d.). The difference in mean Q-T onset between these two groups is significant ($P<0.05$). Among the older subjects, there was no tendency toward increasing Q-T delay with increasing age. Note also that Q EMG onset preceded T onset in 25% of the subjects. This may be related to initial knee position which was not carefully controlled. For example, if the knees of some subjects were slightly flexed prior to the translation, an early Q contraction would hyperextend the knee and pull the lower part of the trunk slightly forward. A previous study (Woollacott et al. 1986) also noted that some subjects had reversed Q-T timing. However, in that study the reversal was only found in their older subjects. Figure 5 shows that Q-T reversal occurred across the entire age range, although there was a slightly larger incidence in older subjects.

Sensory Interaction Tests

As visual and proprioceptive sensory information was removed or made inaccurate during the various sensory test conditions, subjects became less stable (Table 2) and falls became more likely. Figure 6 shows results from three subjects who were attempting to stand as still as possible during the six different sensory conditions. Subject A was typical of most of the population who did not fall in any condition and whose sway amplitudes increased as they were deprived of sensory orientation reference information. Subject B showed average amounts of sway in conditions 1, 2, 4, and 5 but fell in conditions 3 and 6 which both involved sway-referenced vision. Both falls were ballistic since the subject apparently did not attempt to correct the sway trajectory. Subject C showed average sway in conditions 1 through 4 but fell in 5 and 6, in which both proprioceptive and visual sensory signals are altered or absent.

All subjects were very stable, as judged by their peak-to-peak θ_{ap} (Figure 7), with eyes open on a stable surface, and with eyes closed on a stable surface (conditions 1 and 2). These conditions are equivalent to the clinical Standard Romberg test (Black et al. 1982). No subjects fell in conditions 1 and 2. Postural stability decreased in condition 3 when the visual surround rotation was referenced to the subject's sway. The median of the condition 3 sway distribution in Figure 7 was only about one degree higher than condition 2 indicating that most subjects had only slightly more difficulty controlling their posture under the sway-referenced vision condition than with eyes closed. However, the condition 3 distribution is highly skewed toward larger sway amplitudes indicating that a significant fraction of the population had a great deal of difficulty maintaining their upright posture when visual orientation information was present but inaccurate. In addition, 30 of 214 subjects (14.0%) fell on the condition 3.

The condition 4 provided accurate visual cues but inaccurate proprioceptive cues as the support surface upon which the subject stood rotated in proportion to their θ_{ap} sway. On average subjects swayed more under this condition than during conditions 1 and 2. This distribution was skewed toward larger sway angles in a similar manner to the condition 3 distribution. However subjects on average were more stable in condition 4 than in the sway-referenced vision condition since only three subjects out of 214 (1.4%) fell in condition 4.

Visual cues in condition 5 were absent (eyes closed) and proprioceptive cues were inaccurate since the support surface was sway-referenced. This condition presumably forces a greater reliance on vestibular cues for posture control. Average peak-to-peak θ_{ap} was larger than on any of the previous conditions and was also skewed toward larger values. Twenty eight of 214 subjects (13.1%) fell on this condition.

Condition 6 was the most difficult of the six conditions. Under this condition both the visual surround and the support surface were sway-referenced and therefore were providing inaccurate proprioceptive and visual orientation cues. As with condition 5, this condition forced a greater reliance on vestibular cues for posture control. However the presence of inaccurate visual orientation cues in condition 6 (as opposed to absent vision in condition 5) apparently increased the difficulty of the task. The average sway for subjects who completed condition 6 was larger than on any other conditions, and 70 of 214 subjects (32.7%) fell.

Movement Strategy during Sensory Tests

The 65 subjects whose body motions were measured at both the hip and shoulder were the older portion of the entire population with ages ranging from 27 to 81 years (mean 56.2 +/- 12.5 s.d.). The use of an ankle strategy was by far the most common mode of posture control in these subjects. Their mean strategy scores were close to zero and the variances of the scores were small on all six conditions (Table 3). This was confirmed by plotting peak-to-peak θ_{cg} versus peak-to-peak θ_{ap}. For pure hip strategies θ_{cg} and θ_{ap} should be relatively unrelated whereas a pure ankle strategy would have equal θ_{cg} and θ_{ap}. Correlation coefficients between peak-to-peak θ_{cg} and θ_{ap} data ranged from 0.93 to 0.98 for the six conditions. Data points were tightly clustered around the line of equal θ_{cg} and θ_{ap}.

Fall Patterns in Sensory Tests

Table 4 summarizes the data on subjects who fell during one or more of the six conditions. Falls during sensory test conditions were not random occurrences, but rather were associated with the inability of some subjects to obtain and/or coordinate the sensory information available for the control of posture. Consider subjects who fell on two of the four conditions which presented them with sensory conflict situations. There are six possible combinations of paired falls within the grouping of the four more difficult conditions. If paired falls occurred randomly they would be evenly distributed across the six possible combinations. This was clearly not the case since three of the six combinations of paired falls were not observed. That is, no subjects fell on 3-4 and 4-5 paired conditions. Only two subjects fell on the 3-5 and one on the 4-6 combination of conditions. Therefore paired falls were primarily limited to only two of the six possible paired combinations with 12 subjects falling on 5-6 conditions and 15 falling on 3-6 conditions.

The six subjects who fell on three conditions were also not randomly distributed among the 4 possible combinations of the 4 sensory conflict conditions taken 3 at a time. Rather all six subjects fell on the same set of three conditions which was the 3-5-6 combination. This combination is interesting since it combines the features of the two most common paired condition falls, 3-6 and 5-6. Patients which show this pattern of falls have previously been identified (Black and Nashner 1984). These subjects, who constituted only 2.8% of our subjects, could be considered to be quite seriously impaired relative to the remainder of the population. Most of these subjects were older (aged 45, 48, 60, 66, 69, and 70 years).

There was a clear learning effect when sensory tests were repeated immediately following a fall. Thirty-three of the 131 first test falls were repeated. Only 6 of the 33 subjects (18%) fell on the repeated test. The number of repeat test falls for the four sensory conditions where falls occurred were 1 of 8 for condition 3, 0 of 2 for condition 4, 2 of 8 for condition 5, and 3 of 15 for condition 6.

There was evidence of vestibulo-ocular (VOR) and optokinetic reflex (OKR) abnormalities in some subjects who fell on two or more conditions. Of the three subjects with the shortest VOR time constants (Peterka et al. 1989) one was a 5-6 faller and two were 3-5-6 fallers. The 5-6 faller with a short VOR time constant also had a significant partial unilateral loss of vestibular function on the caloric test. The subject who had the largest OKR time delay of any subject tested (268 ms) was also a 5-6 faller. Among subjects over 50 years, two of the three subjects with the lowest OKR gain constants were 3-5-6 fallers, and the other was a 3-6 faller. Finally, the two older subjects with the largest OKR time constants, indicating decreased sensitivity to higher frequency visual field motions, were both 3-6 fallers.

However, VOR and OKR parameters of most subjects who fell on two or more conditions were not distinguishable from those of subjects who did not fall or fell on only one condition. In order to compare the incidence of VOR abnormalities in fallers and nonfallers, the population was divided into two groups based on the number of falls. One group consisted of 36 subjects who fell in two or more different sensory conditions, and the other consisted of

178 subjects who did not fall, or fell in only one condition. The proportion of subjects who had two or more VOR response parameters (based on sinusoidal or pseudorandom testing, see Peterka et al. 1989) on the fringes of the parameter distributions (above or below the upper and lower 2.5 percentile points, respectively) were counted. The proportions for the two groups were nearly identical, with 26.7% for the group with two or more falls, and 27.1% for the other group. Similar results were obtained with OKR data, where 11.8% of the group with 2 or more falls had one or more OKR parameters on the fringes of the parameter distributions, compared to 8.2% for the other group.

Age-Related Changes in Sensory Test Results

Generally, the number of falls increased with increasing age. These results are summarized in Table 5. The incidence of falls was lowest for middle aged subjects (20 to 40 years). Subjects ages 13 to 19 years had a high incidence of single condition falls (33%) but low multiple condition falls (11%). With the exception of condition 3, peak-to-peak sway was also higher in the younger as compared to middle aged subjects (Figure 8). This difference between younger and middle aged subjects was most evident in sensory conditions 4-6 suggesting that younger subjects were sensitive to alterations in proprioceptive cues. The occurrence of single condition falls increased rapidly for subjects older than about 45 years, although the incidence of multiple condition falls remained quite stable through the 50's before showing an increase in the 60 to 70 year olds. A possible anomalous result was obtained in the over 70 age group for multiple falls. Their multiple fall rate was less than the fall rate for 60 to 70 year old subjects and approximately the same as for subjects 40-60 years. This may be a result of the small sample size of the over 70 age group, an exceptionally healthy condition of this group, or alternatively an exceptionally high fall rate for subjects in the 60-70 age group.

The increased incidence of falls in older subjects in conditions 3 and 5 was not accompanied by a trend toward increasing peak-to-peak θ_{ap} among non-fallers (Figure 8). This is in contrast to condition 6 where both sway and falls increased with age. The theoretical limit of peak-to-peak sway is dependent on foot size and body mass distribution since it is not possible to statically position the body's center of gravity outside of its base of support. Since most subjects have a 10 to 12 degree range of stable AP sway, it seems that there was some room for the non-falling population to shift toward larger sways in conditions 3 and 5, and that this shift would be accompanied by increased falls. Although the falls increased, the peak-to-peak sway amplitude of non-fallers did not.

Discussion

Most of the results of motor and sensory tests of postural control showed a wide range of what must be considered normal function. In spite of the large variances, age-related changes in function were evident on many motor and sensory tests. For motor tests, the latency of EMG onsets following support surface translations increased with increasing age. In addition, there was evidence that the rate of increase of EMG onset with age was larger for subjects older than about 55 years. This increased rate was most evident in the tibialis muscle. Studies of muscle strength in the elderly (Whipple et al. 1987) have also shown proportionally larger losses in tibialis strength

compared to other leg muscles. The loss of strength combined with the slowing of the tibialis muscle response to body perturbations would diminish an individual's ability to control backward sway.

Sensory test results showed changes with age similar to motor tests, with most falls occurring in subjects older than about 50 years. Subjects younger than about 15 years had poorer performance on sensory conditions 4, 5, and 6. Since altered proprioceptive postural cues are common to these three sensory conditions, this suggests that younger subjects rely more heavily on proprioceptive cues than do middle aged adults.

Age-related changes in posture control performance were not present in subjects older than about 15 years when they were tested under "normal" operating conditions during sensory test conditions 1 and 2. That is, when subjects stood on an earth fixed support surface with eyes open or closed, their sway was small and the oldest subjects performed as well as the younger ones. Neither the mean sway nor the variance of sway measures showed any age-related trend. This is in agreement with a study by Black et al. (1982), although the age range of their population was smaller. However other studies (Sheldon 1963; Murray et al. 1975) have identified small increases in sway in older subjects under similar conditions. Since our selection criteria excluded any subject with prior indications of balance disorders, our results may be biased in favor of better performing subjects.

Subjects aged 7 to 15 years showed increased peak-to-peak sway amplitudes on all sensory test conditions except condition 3. The increased sway in conditions 1 and 2 is only slightly larger than in adults. This is consistent with previous results (Riach and Hayes 1985) who tested children aged 2 to 15 years and Forssberg and Nashner (1982) in children 1 1/2 to 10 years. Both studies show poorer sensory condition 1 posture performance in the youngest children with a convergence toward adults performance at about age 8 to 10 years. Forssberg and Nashner (1982) also tested their subjects using sway-referenced tests identical to conditions 3 through 6 and again found the poorest performance in the youngest children but with incomplete convergence to adult values by age 10 years. Our results for condition 3 differ from theirs since the average peak-to-peak sway of our youngest subjects did not differ from adult sway values while their young subject performance was poorer than adult performance. However our results in conditions 4 through 6 agree with theirs and extend their findings to show that adult performance is not attained until about age 15 years under these sensory conditions. The only motor control test result which suggested a similar age division at about 15 years was the Q EMG onset time in response to forward support surface translations (Figure 4).

Since the first two sensory tests are characterized by the presence of multiple sensory system inputs which converge and cooperate in the generation of appropriate and accurate posture control responses, it is apparent that subjects are very well adapted for dealing with their environment under normal operating conditions. However it is also clear from other sensory test conditions that the "parts" which make up the "whole" are subject to larger variability. This variability is demonstrated by the wide range of postural sway trajectories recorded under varying conditions of accurate, inaccurate, and absent visual and proprioceptive cues to orientation. In some individuals there was a complete failure to accommodate (resulting in a

fall) to some of the sensory conditions, while in other individuals these same unusual sensory conditions only moderately increased sway compared to the first two sensory conditions.

One could speculate that the posture test sequence was simply too difficult and therefore was overly sensitive to minor deficiencies in sensory system function or in neuromuscular control. This conclusion is not consistent with several aspects of the data. First, there were a large proportion of subjects in any age group who were able to perform all sensory tests with little or no difficulty. Second, many subjects who fell on sensory tests showed no attempt to adjust their posture prior to their fall indicating that their posture control mechanism was nonfunctional under the given sensory conditions for at least several seconds. Third, the shape of the sway distributions in Figure 7, for conditions in which a significant number of subjects fell, show that there was a separation of fallers from the average performance of subjects who did not fall. Fourth, falls on the sensory conditions were not random events but rather occurred in distinct patterns which were the same patterns as those previously shown in patients suffering from vestibular abnormalities (Nashner et al. 1982; Black et al. 1983; Black and Nashner 1984; Black et al. 1988).

A more detailed consideration of the pattern of sway and falls in sensory test conditions 3, 5, and 6 suggests at least two mechanisms which could lead to an increased incidence of falls in the older subjects. First, muscle weakness or delayed responses could reduce the range of peak-to-peak sway amplitudes that fallers could accommodate. Alternatively, the fallers on any given condition could represent a distinct subset of individuals who generate incorrect or absent responses in particular sensory environments. That is, while most subjects produce a graded increase in sway when confronted with a more challenging sensory condition, the subset of fallers appeared to be unable to respond at all, or else responded inappropriately. The second explanation seems more consistent with the results of condition 3. In this condition the peak-to-peak sway of most subjects was far below the presumed fall threshold. In contrast, the first explanation fits the results from condition 5 in which peak-to-peak sways were closer to the fall threshold. Both factors may be present in condition 6. That is, since subjects are swaying more on average in condition 6, an increased number could sway past their threshold and fall as the fall threshold declines with increasing age. In addition, based on the results in condition 3, a subset of the population consisting mostly of older subjects could respond inappropriately to the sway-referenced visual surround condition and fail to generate timely posture corrections.

The two paired fall combinations which produced the majority of the paired falls can be logically associated with specific types of peripheral sensory or sensory integration problems. The 15 subjects who fell on the 3-6 conditions were highly dependent on vision for their orientation reference. These subjects behaved paradoxically. The fact that they did not fall in condition 5 indicates that they had sufficient vestibular function to properly maintain stable stance using no visual and inaccurate proprioceptive sensory cues. However when visual cues were present in 3 and 6, they chose to ignore accurate vestibular and proprioceptive cues in condition 3 and accurate vestibular cues in condition 6 in favor of the inaccurate visual reference.

The second most common paired fall conditions (5 and 6) forced subjects to rely primarily on their vestibular systems for posture control since proprioceptive and/or visual cues are either absent or inaccurate in both conditions. Subjects who fell on these conditions either had a vestibular deficit or were not able to use vestibular information for postural control. A subject with total bilateral peripheral loss of vestibular function is the extreme form of vestibular deficiency. Patients with bilateral loss as judged from absent caloric and rotation responses invariably fall in conditions 5 and 6 (Nashner et al. 1982; Black et al. 1983). Since independent tests of vertical canal and otolith function (e.g. vertical plane rotations or ocular counterrolling) were not performed, the possibility that these paired fall subjects had absent or abnormal vertical canal or otolith function cannot be ruled out. However if vertical canal or otolith function were abnormal in these subjects, the peripheral abnormality was not distributed throughout the entire vestibular organ since rotation and caloric tests, which primarily stimulate the horizontal semicircular canals, were normal in most subjects.

The pattern of falls in conditions 5 and 6 could theoretically also arise from central mechanisms. The central postural control mechanisms perform complex tasks which include both the selection of the appropriate sensory orientation reference in the face of conflicting cues from several sensory systems and the generation of the correct motor commands to the muscles. It is possible that peripheral vestibular signals may be normal, but central mechanisms which make use of this information are faulty. The "fault" may have more than one source. For example, the processing of the sensory information may simply be too slow, in which case the appropriate motor commands never arrive at the muscles or arrive too late to prevent a fall. Alternatively, the central processing which must deal with conflicting sensory information may produce inappropriate responses based on the available sensory signals. These inappropriate responses could drive the system into instability with a resulting fall.

Finally, subjects with weak muscles relative to their body masses would be more susceptible to falls than stronger subjects since the lower forces generated by weaker subjects would diminish their ability to correct for postural perturbations. This effect would be particularly important near the limits of stability where maximal forces are necessary to move the subject away from the brink of a fall. Since conditions 5 and 6 are the most difficult in the sense that they bring subjects closer to the threshold of a fall than do the other four conditions, the relative muscle strength of subjects would be more likely to play a role in the last two conditions.

The large number of falls in sensory test conditions probably reflects a combination of sensory system deficits and inappropriate central nervous system coordination of sensory information. There was no evidence of proprioceptive system deficits in fallers vs. non-fallers based on EMG onset times following translations. The major source of medium and long latency EMG responses is probably muscle spindles (see Brooks 1986 for a review) with little or no contribution from ankle joint afferents or skin pressure receptors (Diener et al. 1984), although there may be some vestibular contribution (Allum 1983). However there were a number of subjects who fell on two or more conditions who also had VOR and OKR response parameters which were on the tails of the population distributions of those parameters. Since

it is reasonable to assume that distance from the mean reflects abnormality, there was at least a partial correlation between subjects with abnormal VOR and OKR results and abnormal posture control results. The correlation was not perfect, since most subjects who fell on two or more conditions showed no VOR or OKR abnormality. There are at least three possible explanations for the weak correlation between VOR and OKR abnormalities and poor postural control. First, our VOR tests measured primarily horizontal canal function, whereas head movements during posture testing primarily stimulate vertical canals and otoliths. To the extent that a vestibular abnormality could only affect one or a limited number of the vestibular receptors in each ear, horizontal VOR and posture results could be different. Second, our OKR tests characterized the optokinetic reflex for motions of the visual field in the horizontal plane. However, the visual system contribution to posture control during our sensory tests would be associated with the detection of pitch plane movement and with depth cues from the disparity of images on the retina of each eye. Pitch plane OKR and vergence control responses might be more highly correlated with posture control deficits. Third, differences between our VOR and posture test results could relate to central nervous system problems in the organization of sensory system interactions. That is, sensory system signals and vestibulo-ocular reflexes could be normal in some subjects with abnormal posture test results if the postural abnormality were due to their inability to effectively deal with conflicting information from vestibular, proprioceptive, and visual systems.

It would be reasonable to conclude that approximately 10 to 15% of this screened population had significant posture control deficits. The deficits in a few subjects were clearly traceable to vestibular deficits, e.g. one subject with an asymptomatic unilateral loss of vestibular function. Others had rotation test results which were suggestive of peripheral vestibular problems. However since the role of the central nervous system in the coordination of sensory signals from multiple systems is poorly understood, it is not possible to rule out the contribution of central interaction problems as opposed to peripheral sensory problems. It is apparent that equilibrium control deficits exist in a putatively normal population, these deficits are more common in older subjects, but are normally masked by the presence of redundant sources of sensory orientation cues. In susceptible subjects, the loss of redundant information can unmask their deficit resulting in a sudden loss of postural control.

Acknowledgment. We wish to thank Monika Schoenhoff, Christopher Newell, Patrick Shea, and Martha Benolken for their assistance, and Drs. Charlotte Shupert and Alar Mirka for insightful comments. This research was supported by NASA grants NCC9-8 and NAG 9-117, and NIH grant NS-19222.

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Table 1 - Sensory test conditions

		TEST CONDITIONS		SENSORY CONDITIONS	
Condition	Sensory Conflict	Visual Reference	Support Surface Reference	Accurate	Inaccurate
1	no	earth	earth	vis, vest, prop	
2	no	eyes closed	earth	vest, prop	
3	yes	sway	earth	vest, prop	vis
4	yes	earth	sway	vest, vis	prop
5	yes	eyes closed	sway	vest	prop
6	yes	sway	sway	vest	prop, vis

Table 2 - AP Sway Measures on Completed Sensory Test Conditions
All values are in degrees (mean +/- 1 s.d.)

Condition	N	Average Rectified θ_{ap} Sway	Peak-to-Peak θ_{ap} Sway
1	214	0.26 +/- 0.21	0.82 +/- 0.44
2	214	0.42 +/- 0.33	1.25 +/- 0.58
3	189	0.68 +/- 0.57	2.78 +/- 2.00
4	213	0.74 +/- 0.49	2.95 +/- 1.97
5	192	1.20 +/- 0.48	5.54 +/- 2.23
6	155	1.32 +/- 0.53	5.75 +/- 2.12

Table 3 - Completed Sensory Test Sway Measures for Subjects with Sway Measured at Shoulder and Hip.
All values are in degrees (mean +/- 1 s.d.)

Condition	N	Peak-to-Peak θ_{ap} Sway	Peak-to-Peak θ_{cg} Sway	Peak-to-Peak θ_{h} Sway	Strategy Score
1	65	0.75 +/- 0.32	0.72 +/- 0.34	1.41 +/- 0.56	-0.01 +/- 0.05
2	65	1.21 +/- 0.54	1.33 +/- 0.59	1.78 +/- 0.81	0.04 +/- 0.07
3	46	3.01 +/- 2.16	3.28 +/- 2.18	3.56 +/- 2.09	0.24 +/- 0.56
4	65	2.41 +/- 1.20	2.22 +/- 1.15	2.96 +/- 2.10	-0.14 +/- 0.46
5	56	5.31 +/- 2.45	5.50 +/- 2.37	5.53 +/- 3.44	0.21 +/- 1.38
6	35	5.27 +/- 1.86	5.58 +/- 2.12	6.08 +/- 4.91	0.49 +/- 0.95

Table 4 - Falls on Sensory Test Conditions

	Conditions	# Subjects	% of Total N = 214
1 Fall			
	6	36	16.8%
	5	8	3.7%
	3	7	3.3%
	4	2	0.9%
		53	24.8%
2 Falls			
	3,6	15	7.0%
	5,6	12	5.6%
	3,5	2	0.9%
	4,6	1	0.5%
		30	14.0%
3 Falls			
	3,5,6	6	2.8%
1, 2, or 3 Falls		89	41.6%

Table 5 - Sensory Test Falls Sorted by Subject Age

Age Group	#Subjects	Single Falls N	Single Falls %	Multiple Falls N	Multiple Falls %	Total Falls N	Total Falls %
7 - 12	21	3	14.3%	2	9.5%	5	23.8%
13 - 19	27	9	33.3%	3	11.1%	12	44.4%
20 - 29	28	4	14.3%	2	7.1%	6	21.4%
30 - 39	32	4	12.5%	2	6.3%	6	18.8%
40 - 49	32	9	28.1%	6	18.8%	15	46.9%
50 - 59	26	8	30.8%	5	19.2%	13	50.0%
60 - 69	35	10	28.6%	14	40.0%	24	68.6%
70 and over	13	6	46.2%	2	15.2%	8	61.5%
Total	214	53	24.8%	36	16.8%	89	41.6%

Figure Legends

Figure 1. Definition of body angles for AP sway in a sagittal plane.

Figure 2. EMG responses of one individual to support surface translations. EMG responses to each of five individual trials (thin lines) and the average (thick line) are shown for backward translations causing forward body sway (left column) and forward translations causing backward body sway (right column). Arrows indicate the onset times (in ms) of EMG bursts following the start of translations. Onset times were estimated from the average EMG traces.

Figure 3. Relation between EMG onset times of proximal and distal leg muscle responses to backward support surface translations causing forward sway (left plot, N=102) and forward translations causing backward sway (right plot, N=118). Solid lines through data are lowess fits. Dashed lines represent equal proximal and distal muscle EMG onset times.

Figure 4. EMG onset times from support surface translations as a function of subject age. Plots are based on recordings from 163, 122, 198, and 119 subjects for G, H, T, and Q, respectively. Solid lines through data are lowess fits.

Figure 5. Differences between the EMG onset times of proximal and distal muscle responses to support surface translations as a function of subject age. Plots are based on recordings from 102 and 118 subjects for H-G and Q-T, respectively. Solid lines through data are lowess fits.

Figure 6. Sensory interaction test results showing θ_{ap} sway of three subjects during six sensory test conditions. Subject ages were 31, 61, and 67 years for A, B, and C respectively. See methods section for the description of the sensory test conditions.

Figure 7. Histograms of peak-to-peak θ_{ap} under the six different sensory test conditions. Gray bars to the right of each histogram indicate the number of subjects who fell in that condition. One subject in condition 3, and 5 subjects in condition 5 had sways greater than 12^{o} and did not fall, and are not included in those histograms.

Figure 8. Peak-to-peak θ_{ap} as a function of age for the six sensory test conditions. Solid dots at the top of each graph indicate subjects who fell in that condition. Solid lines are lowess fits to the data for subjects who did not fall during the test.

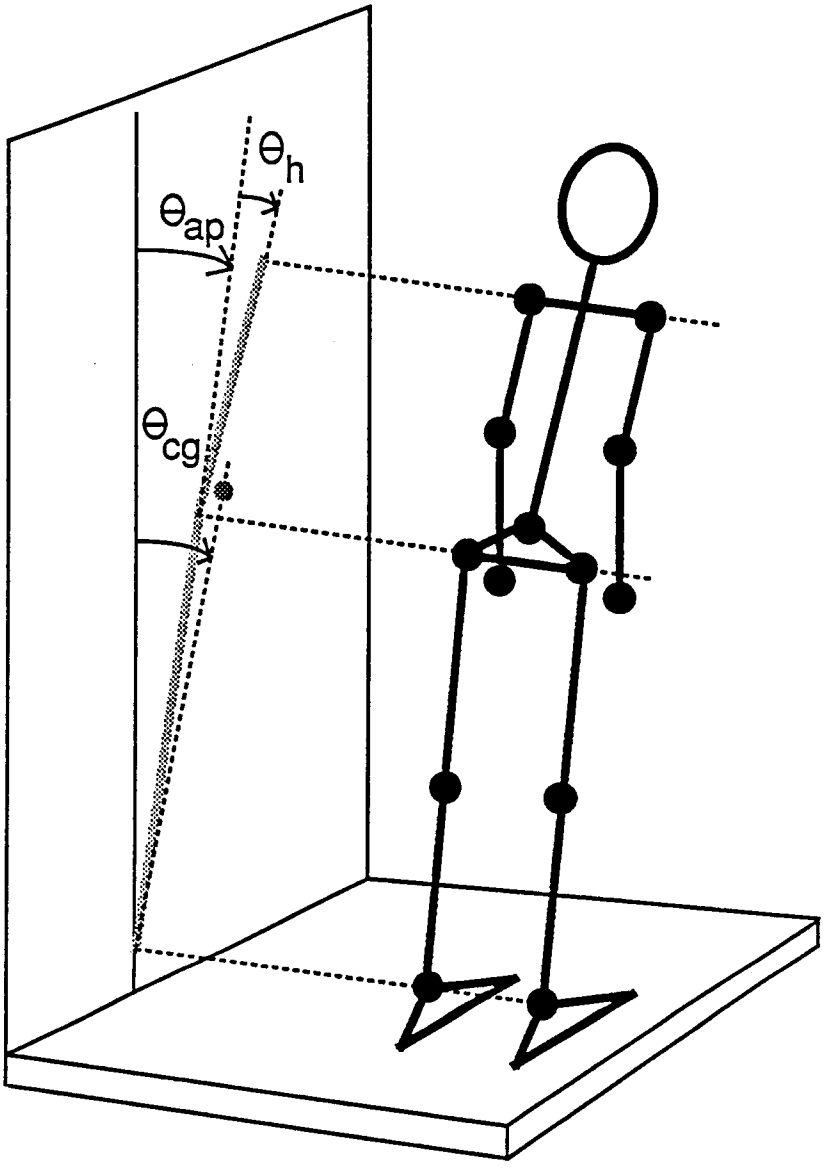


Figure 1

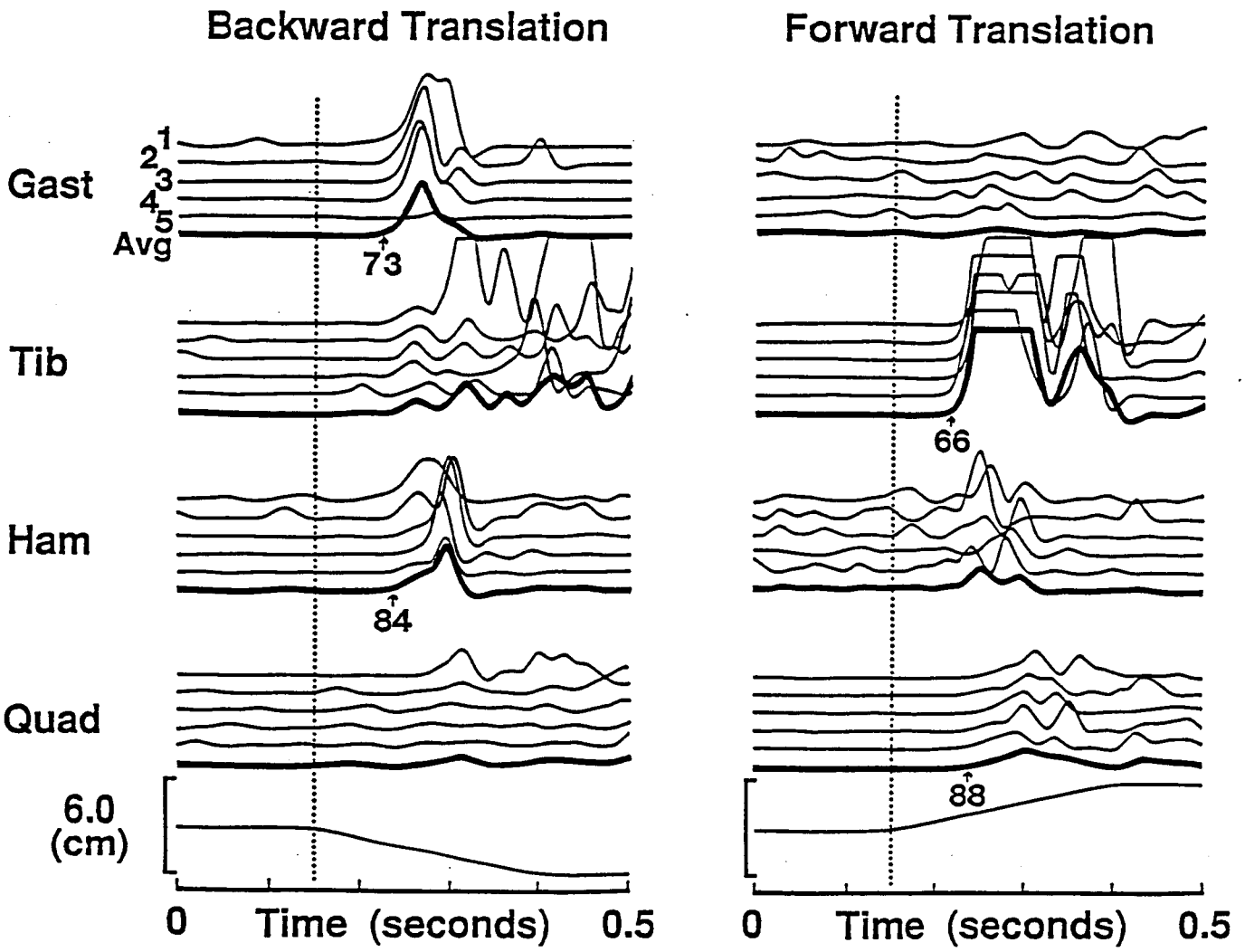
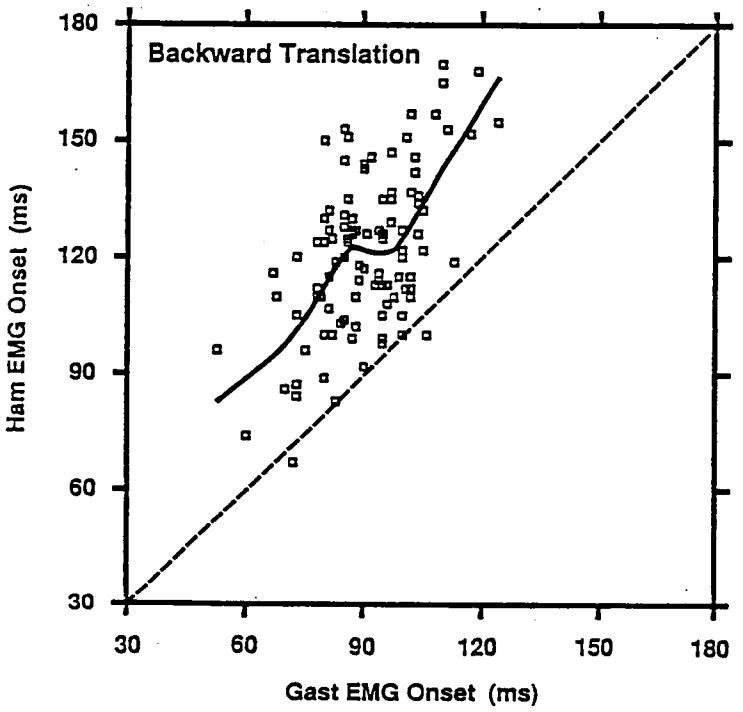

Backward Translation
Forward Translation
Gast
1
2
3
4
5
Avg
73
Tib
66
Ham
84
Quad
88
6.0
(cm)
0
Time (seconds)
0.5
0
Time (seconds)
0.5

Figure 2


Backward Translation
Ham EMG Onset (ms)
Gast EMG Onset (ms)
180
150
120
90
60
30
30
60
90
120
150
180

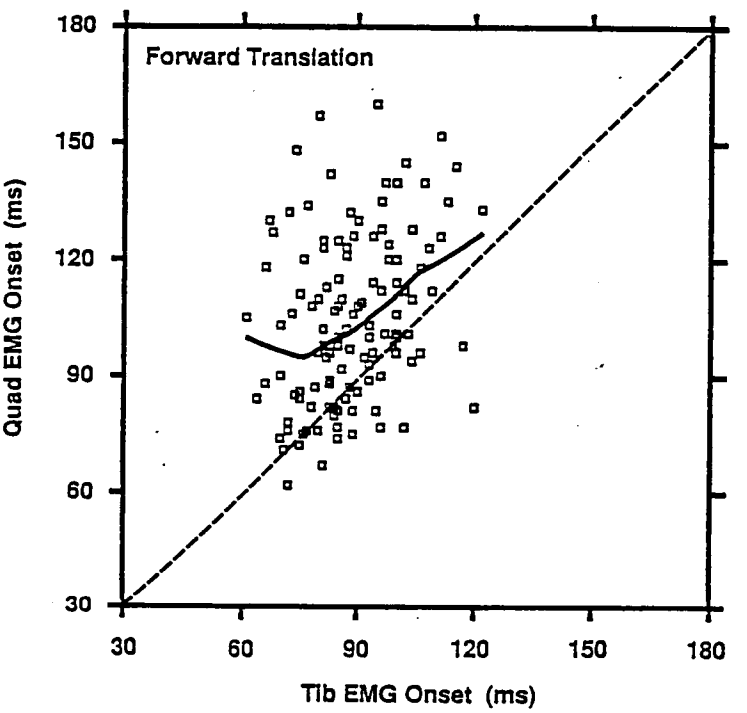

Forward Translation
Quad EMG Onset (ms)
Tib EMG Onset (ms)
180
150
120
90
60
30
30
60
90
120
150
180

Figure 3

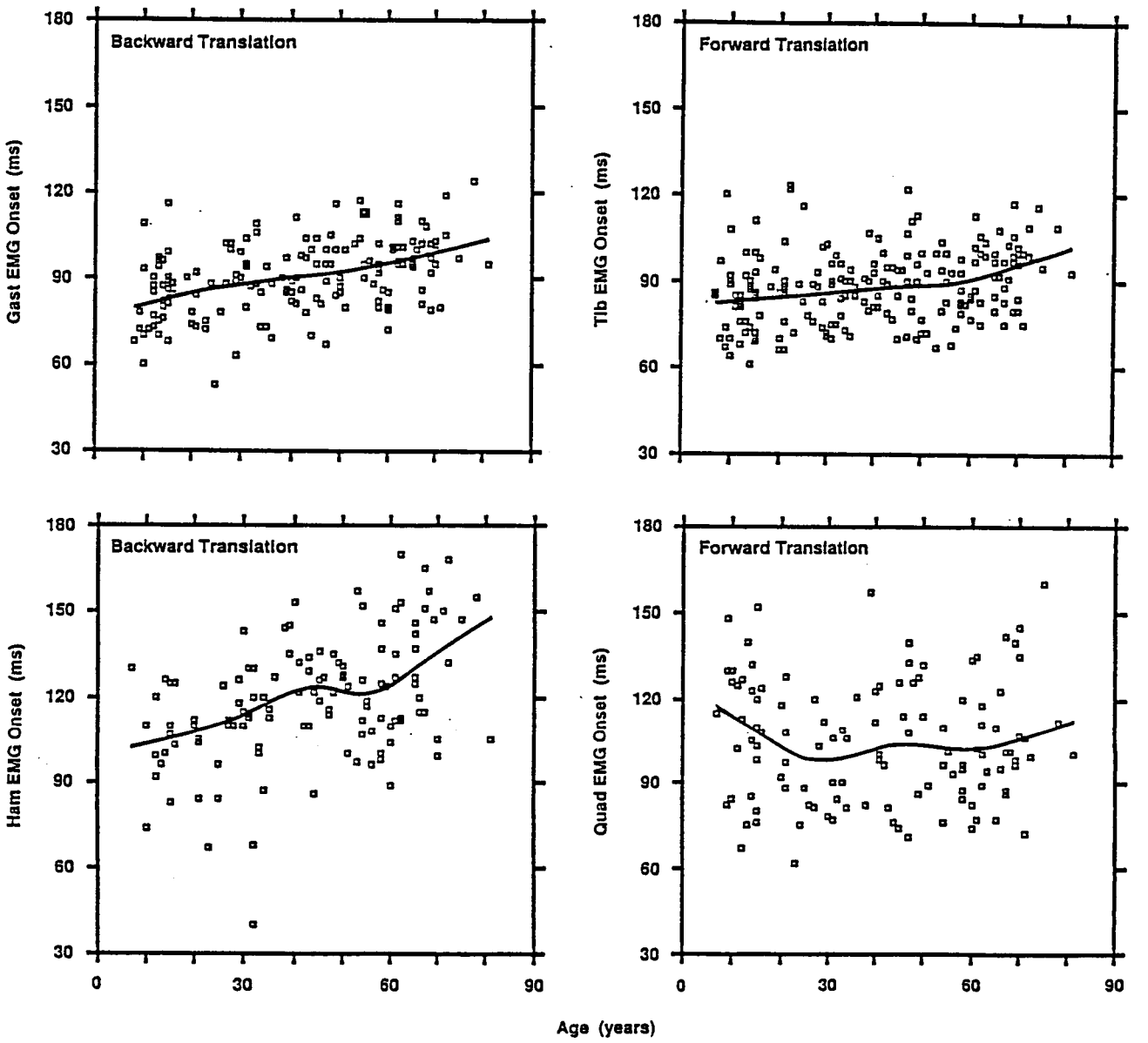

Backward Translation
Forward Translation
Backward Translation
Forward Translation
Gast EMG Onset (ms)
Tib EMG Onset (ms)
Ham EMG Onset (ms)
Quad EMG Onset (ms)
180
150
120
90
60
30
0
30
60
90
Age (years)

Figure 4

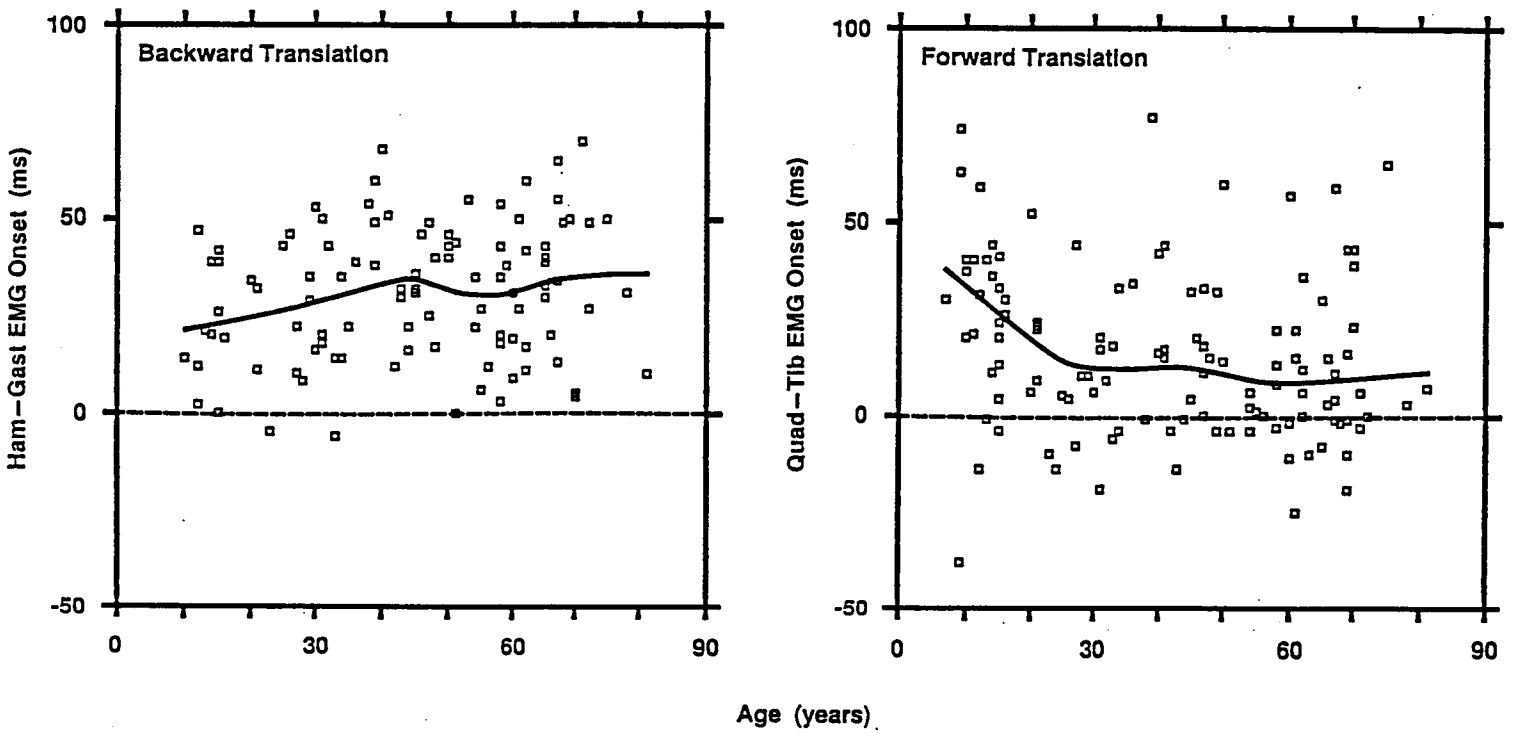

Backward Translation
Forward Translation
Ham−Gast EMG Onset (ms)
Quad−Tib EMG Onset (ms)
100
50
0
-50
0
30
60
90
Age (years)

Figure 5

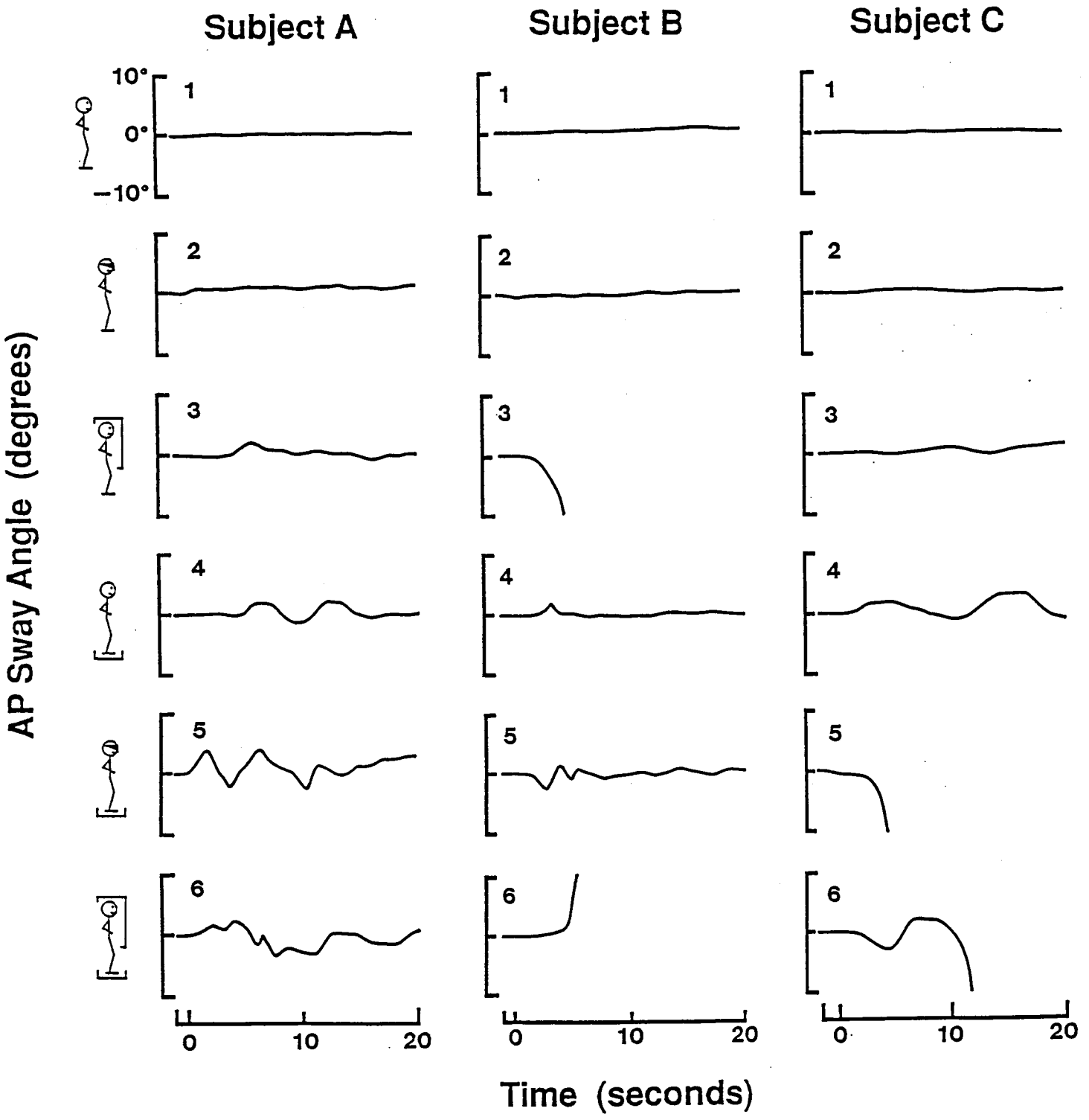

Subject A
Subject B
Subject C
AP Sway Angle (degrees)
10°
0°
−10°
1
2
3
4
5
6
0
10
20
Time (seconds)

Figure 6

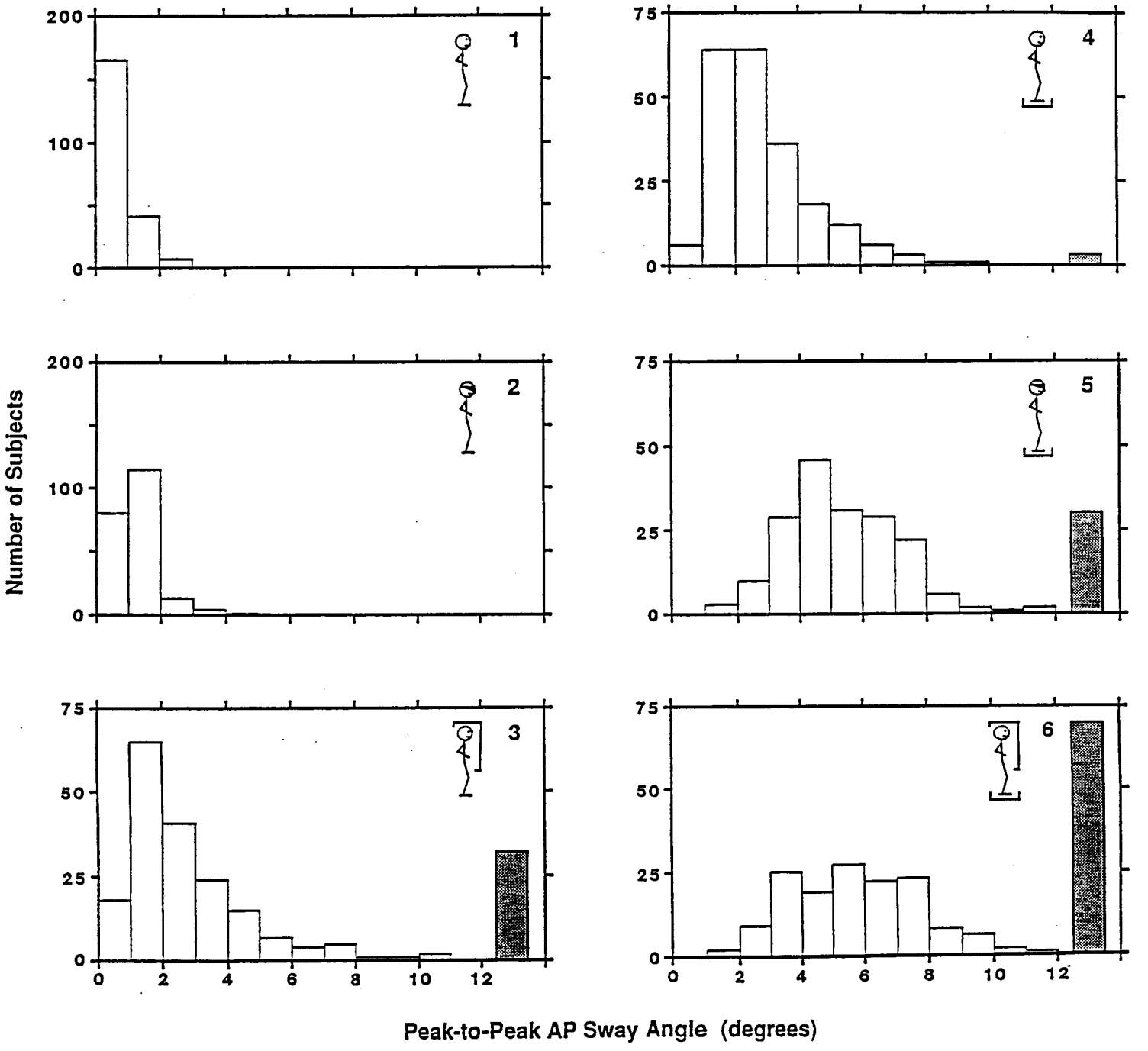

Number of Subjects
Peak-to-Peak AP Sway Angle (degrees)
1
2
3
4
5
6
200
100
0
75
50
25
0
2
4
6
8
10
12

Figure 7

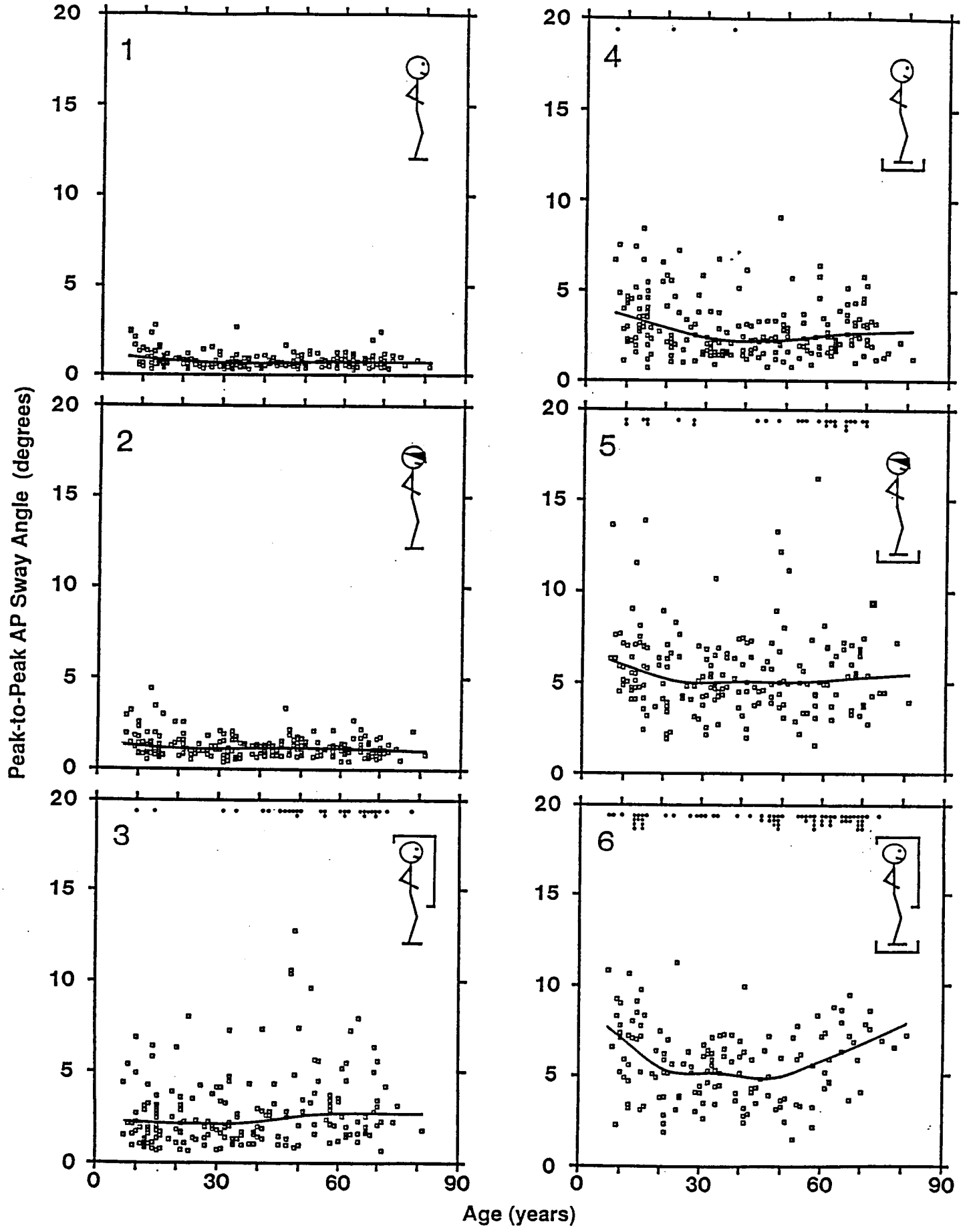

1
2
3
4
5
6
Peak-to-Peak AP Sway Angle (degrees)
Age (years)
0
5
10
15
20
0
30
60
90

Figure 8